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CONTENTS

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	PAGE
Auricular Fibrillation — *Spencer A. Folsom, M.D., Orlando.*	11
Insomnia — *H. Mason Smith, M.D., Tampa.*	18
Artificial Pneumothorax in the Treatment of Pulmonary Tuberculosis — *M. J. Flipse, M.D., Miami.*	22
Spinal Anesthesia — *John R. Boling, M.D., F.A.C.S., Tampa.*	27
Caudal Anesthesia in Rectal Surgery — *C. Larimore Perry, M.D., Miami.*	30
Editorials: (1) Get What It's Worth; (2) County Societies	32, 33
State News Items	33-36
Proceedings of the Eleventh Annual Meeting of the Florida Railway Surgeons' Association	36
Schedule of Meetings—Component Societies	37
Woman's Auxiliary Page	38
Tuberculosis Abstracts	40-46
Index to Advertisements	40

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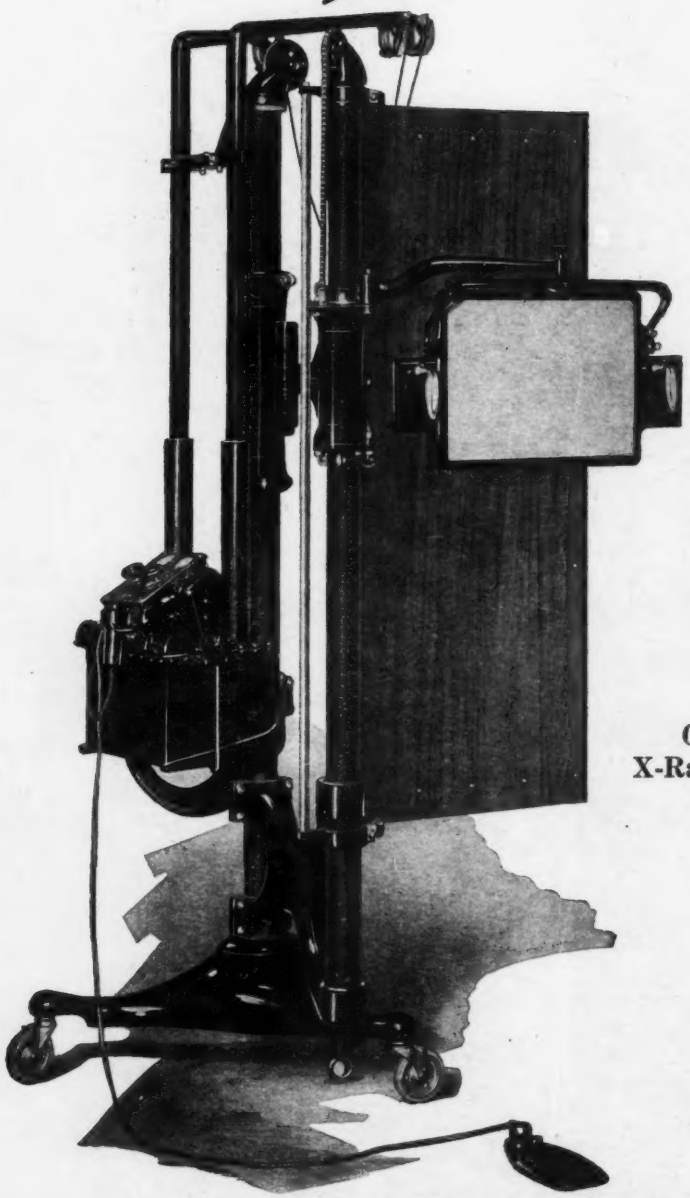

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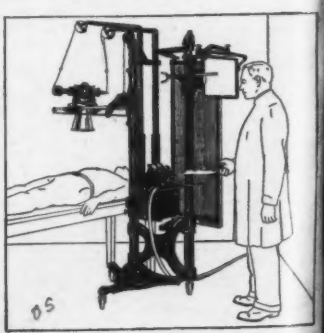

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AURICULAR FIBRILLATION*

SPENCER A. FOLSOM, M.D.,
Orlando.

"Ignorance is the night of the mind, but a night without moon or star."
—Confucius.

About three decades ago, the night of the mind really existed for those indomitable searchers who falteringly and hesitatingly groped in the darkness of ignorance for the light of knowledge. The light was elusive, but was found after careful, patient, painstaking effort. It burns today, but not as brightly as we would have it because the darkness approaches too closely the circle of its illumination. We still need more light.

I cannot repress my envy of the men who have been so fortunate as to have lived and practiced all these years and seen the wonderful and surprising changes of thought take place with kaleidoscopic rapidity. This irregularity of the heart's action held a fascinating interest for them, and it was with a full measure of satisfaction, such as we younger men can hardly appreciate, that they saw the mystery solved and cosmos brought out of chaos. It hardly matters whether we call this condition a clinical entity or a symptom-complex. I should judge that it is entirely correct to call it either or both.

The first work on this subject was done by Mackenzie in the year 1890 when he studied the irregularity of the pulse. Previous to 1890 he began using the jugular as well as the radial tracings in the study of the heart's action. I might mention, at this juncture, that Riegel, for instance, had made such observations previously but Mackenzie was the first to use the method systematically.

Mackenzie, in studying cardiac irregularities, tried to separate the great majority of them into groups and conceived the idea that records of the jugular vein might give light on the significance of these cardiac irregularities. There are three waves of the normal jugular pulse obtained by the polygraph. These waves are the a, c, and v. The a wave is due to contraction of the auricle. There is some difference of opinion as to the causation of the c wave but Mackenzie believes it possibly due to closure of the semilunar valves. Whatever the actual causation, it occurs during the contraction of the ventricle. To summarize, the a wave is due to contraction of the auricle; the c wave to the contraction of the ventricle; the interval, that is, the a to c interval, is one-fifth of a second. The v wave we believe to be due to the closure of the tricuspid valve plus stasis.

Mackenzie found that in 60 to 70 per cent of the cases of irregularity that the a wave was absent. He called such a venous tracing the ventricular form of venous pulse because he thought the auricle and ventricle contracted simultaneously. The idea was that the auricular contraction disappeared and only the ventricular contraction took place. This form of irregularity, with absence of the a wave in the jugular tracing, was called the "nodal rhythm."

In the year 1902 Mackenzie published a very unusual and remarkable book entitled "Study of the Pulse." This work stirred the world of medicine. However, before the appearance of the book definite progress had been made in the the differentiation of the cardiac irregularities. Nearly all the important facts relative to heart-block had been worked out and the extra-systolic arrhythmias were fully understood. This curious irregularity, however, remained shrouded in mystery and obscurity.

In 1903 Einthoven published his epoch-making discovery of an unusually delicate galvanometer. With this instrument, by suspending a very delicate thread of silvered quartz in a magnetic field he was able to register accurately deflections caused by the very faintest electric current. Clinical reports obtained by using this machine appeared in the literature in 1907.

The use of the electrocardiograph in clinical work in the irregularities of the heart brought to light their mechanism of production. Previous to this they were recognized clinically, but here knowledge stopped.

*Read before the 57th Annual Meeting of the Florida Medical Association, Pensacola, May 6, 7, 1930.

Since auricular fibrillation was not known the clinicians were wont to give vent to various fanciful names to the disordered irregular pulse. Thus into medical writing came "the absolutely irregular pulse," "the pulsus arrhythmicus," "the mitral pulse," "pulsus irregularis perpetuus," "completely disordered pulse," the "P-I-P" pulse, "the grossly irregular pulse," "perpetual arrhythmia," "delirium cordis," etc. The word perpetual, as oft-repeated in the various terms given to the irregularity is significant. It was believed then that the irregularity was really perpetual. In a way they were correct but recent knowledge also shows that they were more incorrect.

Lewis, in London, 1909[1], and Rothberger and Winterberg in Vienna, showed conclusively with the electrocardiograph that the mysterious irregularity of the heart was caused by fibrillation or trembling of the auricles. Previous to this the possibility had been suggested by Cushny, 1906[2], Mackenzie[3], Wenckebach[4], and Herring. Thus the erroneous "nodal rhythm" as described by Mackenzie in his jugular tracings was found by Lewis, with the aid of the electrocardiograph, to be auricular fibrillation.

The familiar saying in medicine, so well known to all clinicians, "Know syphilis in all its manifestations and you know medicine," might very appropriately be applied to this irregularity because if one knows auricular fibrillation and knows it well he knows the arrhythmias.

The great frequency of its occurrence renders it imperative that all of us should become very familiar with it; for 60 to 70 per cent of all cases of serious heart failure met with in practice owe the failure directly to this condition, or have the failure aggravated by its presence.[5] When we recognize the various signs and symptoms, they afford us grounds for a sure diagnosis, a safe prognosis, and for a rational line of therapy in a large proportion of cases of cardiac insufficiency.

What is auricular fibrillation? The exact condition bringing about this symptom and sign complex is shown by the electrocardiogram to be mainly in the auricle. The auricular muscle fibers cease to contract rhythmically in response to the contraction impulses which come to them from the sino-auricular node, the pace-maker of the heart. Contractions of individual auricular fibers occur, but each fiber contracts at its rhythm independently of the other fibers. This results in a momentary contractile movement in one part of the auricle, neutralized by a momentary diastole in other parts of the organ. The net result of this disorder is a distended, dilated auricle standing in trembling diastole.

The disturbance in the organ is due to myocardial degeneration(?) and to what has been called a "circus impulse." A normal contraction impulse is sent into the auricle by the sino-auricular node. Instead of being conducted in an orderly manner through the walls of the auricle to the auricular-ventricular node (junctional tissue), the impulse is diverted from its normal course by degeneration within the auricular walls, which block the normal impulse paths. The contraction impulses circulate in the auricular walls, being shunted here and there, and in an irregular manner are picked up by the auricular-ventricular node, which conveys the impulse to the bundle of His, which passes the impulse on to the ventricles. The ventricles are incited to irregular tumultuous activity. What is the result of the abnormal ventricular activity upon the systemic circulation? Some of the ventricular contractions are so weak that they cannot open the aortic valves and therefore cannot produce corresponding pulse waves in the radials. Another reason for the non-opening of the aortic valves may be that the ventricular systoles, otherwise sufficiently strong, do not properly direct the blood toward the aortic valves. In either case it is apparent that there will be more heart-beats than pulse-beats. The stronger ventricular systoles open the aortic valves in the usual fashion and produce correspondingly strong pulse beats. But since even these effective ventricular systoles vary in force and rhythm there is a corresponding variation in the propagated arterial waves as shown by a pulse which is also irregular in force and rhythm. Why do patients with auricular fibrillation so frequently decompensate? The reason depends primarily on the resultant irregular ventricular activity. When the latter is present the myocardium becomes exhausted by the persistent rapid rate and the blood cannot be thrown into the systemic circulation in an orderly fashion.[6]

In auricular fibrillation the degree of functional A-V block influences both the clinical picture and the treatment. The greater the block, the slower the ventricular rate.

Some of the conditions inducing auricular fibrillation are well known to every one, others are obscure, indefinite and indeterminable. Unusual etiologic factors are being reported often in the literature.

Experimentally, fibrillation of the auricles can be produced by stimulation of the vagus, stimulation of the sympathetic, direct faradization of the auricles and injection of thyroid extract intravenously. (One experiment.)

Clinically, it is seen most frequently in rheumatic endocarditis with the valvular lesion of mitral stenosis. Lewis well states that auricular fibrillation and mitral stenosis are "bosom companions." Uncomplicated aortic insufficiency very rarely co-exists with auricular fibrillation.[7] Next in frequency is cardiosclerosis, which is a generic term including gross pathologic changes in all the cardiac structure, valves, endocardium, myocardium, aorta and coronaries. Hypertension or cardiac insufficiency may or may not be present. Lastly, is the so-called extra-cardiac group which is smallest in number.

Some make bold to state that auricular fibrillation cannot exist without an affected myocardium. They doubt the existence of auricular fibrillation occurring in a normal heart. This is a question that is undecided at the present time.

Approximately 66 per cent of all fibrillators belong to the so-called rheumatic group which is preponderantly composed of women 15 to 40 years of age and usually in women suffering from mitral stenosis. (Fifty-two per cent.)

The non-rheumatic group is most frequent in men 45 to 70 years of age.[8] This is the cardiosclerosis group. In both of these groups the fibrillation is usually more or less permanent.

Auricular fibrillation is no respecter of age. Resnik and Scott have recently reported the youngest case of auricular fibrillation on record, aged four years. Previously four children of ten years or less with this affection had been reported.

The extra-cardiac causes in patients with normal and abnormal hearts may be listed as follows: in many of the acute infections such as pneumonia, diphtheria, typhoid, paratyphoid, severe tonsillitis, acute cholecystitis, acute appendicitis, scarlet fever, acute endocarditis and acute pericarditis. Fibrillation is usually transient in these infections.

Felberbaum and Finesilver have reported the occurrence of transient fibrillation in six cases of abdominal disease, in three at the time of gallstone attacks, in one associated with gastric ulcer symptoms and signs, and one with acute appendicitis relieved by operation and in one associated with extensive post-operative adhesions.[9]

Tobacco, alcohol, digitalis, coffee and sulphuretted hydrogen can cause auricular fibrillation. Canby Robinson cites a case of his own that seems to afford evidence that auricular fibrillation may be set up in a healthy man by means of a wholly extra-cardiac factor. This case was a man overcome with the fumes of hydrogen sulphide. Recovered in three hours from the fibrillation. Left the hospital in three days. Reported later as well in every way.[10] Reid reports a case of auricular fibrillation which persisted for two years and eight months. No return of fibrillation since its disappearance ten months ago.[11] Digitalis can and does induce auricular fibrillation in predisposed cases or the fibrillation is brought on by toxic amounts of digitalis in association with other signs of digitalis poisoning. Mackenzie states that the digitalis may induce the fibrillation in cases of cardiac decompensation with slow heart rates.

Occurring in the course of surgical anesthesia or following shortly after surgical operations we sometimes have a transient form of fibrillation. In thyroid disease the condition occurs with about equal frequency in both adenoma and hyperthyroidism. Here the fibrillation occurs permanently or in paroxysmal attacks. It is well that an over-junctioning thyroid be ruled out as a cause in every case of paroxysmal fibrillation of the auricles.

Other etiologic factors that are mentioned are fright, fear, trauma, excitement and effort. Hays and Jones have recorded five cases of auricular fibrillation trauma, in one case a powerful electric shock and in the other four sudden unexpected physical strain. In all but one of the cases the hearts were apparently normal.[12]

Lastly, we must mention the attacks of so-called paroxysmal fibrillation of unknown origin either in men or women, probably heralding the onset of temporary or permanent heart muscle failure; often associated with paroxysmal dyspnea, periodic vertigo and nausea, attacks usually disappearing promptly with rest in bed; often resulting (as many believe), after many recurrences, in permanent fibrillation.[13]

The pathology of the condition is not understood as no distinctive lesion or lesions have been found. What brings the fibrillation on and what are the pathologic changes in the auricular muscle? Both of these questions have not been answered satisfactorily.

Two general possibilities are admitted in regard to the first question. First, that there is some

pathologic change in the muscle fiber of the atria which renders them prone to fibrillate so that some sudden strain or the cumulative effect of the prolonged overwork, such as might occur in valvular disease, is sufficient to initiate the process. The second possibility is that some extrinsic stimulus, such as an alteration of the blood supply to the atria or a change in the chemical composition of the blood itself, would, if sufficiently marked, cause fibrillation in a previously healthy heart and still more readily in one already diseased. Canby Robinson points out that clinical experience is in favor of the first possibility because auricular fibrillation usually occurs in patients with cardiac lesions that are clinically demonstrable, especially with such lesions as throw an extra strain upon the atrium. Nevertheless, cases have been observed in which fibrillation took place in apparently healthy hearts as has been already mentioned. Mackenzie states: "In the hearts which I have examined, which showed auricular fibrillation during life there has been found in the auricle and ventricle, an increase of fibrous tissue and of nucleated cells in the muscular walls. . . . At present, we can only say that one predisposing condition is certain organic changes in the muscle walls of the auricle."[14]

Frothingham reports a study of 11 cases of auricular fibrillation and 23 cases with normal rhythm, at post-morten examination, with especial reference to auricular pathology. No typical lesion was found. There were slight degenerative changes in the muscle fibers of the auricle in fibrillating cases more often than in cases with normal rhythm.[15]

Neuhof mentions as some abnormal factors found in or productive of auricular fibrillation in the human heart the following: enlarged and dilated auricles, pathologic changes in the pacemaker (sino-auricular node), and changes in the nerve tone in the pace-maker from destruction of nerves, fibrils and ganglia and from abnormal impulses reaching the pace-maker.[16]

At the present time our true knowledge of the pathology of auricular fibrillation is very deficient. We tentatively say that a fibrillation in this or that condition is permanent, transient or paroxysmal, but we do not know why. The fibrillation may occur abruptly once and become permanent. More frequently, however, it occurs and disappears, then recurs and recurs, and finally becomes permanent.

Miller has brought out the fact that if the cardiac rhythm is altered to produce auricular fibrillation sub-acute bacterial endocarditis does not occur, despite the fact that the patient may have had for a long time a heart damaged and made vulnerable by rheumatism, syphilis, arteriosclerosis or hyperthyroidism. This strange observation has been corroborated by many clinicians. It is, therefore, pertinent to state that a negative blood culture is the rule in almost any situation where an auricular fibrillation appears.

We do not know if the blood develops any specific bactericidal properties or whether organisms cannot remain in the circulation because of a faulty mechanical pump, or whether subacute bacterial endocarditis in some peculiar form, for example, does not occur here after all, but stays undetected.[17]

It is important to be able to interpret the symptoms and signs of auricular fibrillation because of its predominating frequency compared to the other arrhythmias. Also, it is easier to grasp and comprehend the other irregularities if this is understood. Another reason is that it is the one arrhythmia which may be benefited most often therapeutically.

Symptomatically, auricular fibrillation may be latent, or similar to other cardiac affections. Although rapid pulse is most frequently complained of we must not lose sight of the fact that a slow pulse may be present. Again a regular ventricular rhythm may be present.[18] There are two types of patients present themselves for diagnosis, first, those with good compensation who may complain of rapid pulse, palpitation and dyspnea. They will describe the palpitation as a "thumping of the heart" due to the irregularity. The second and most common type is the one with broken compensation who presents himself complaining of dyspnea, precordial pain, palpitation, cough, etc., and has the objective signs of cyanosis, edema of the feet and ankles, etc. Some patients complain of gastric distress, a sense of fullness, bloating and of tightness, which is apt to focus the attention both of the physician and the patient upon the stomach rather than upon the heart. In every middle-aged patient complaining of gastric symptoms, especially if there is dyspnea, one should think of the heart.

The differentiation and recognition of auricular fibrillation can usually be made at the bedside by a study of the pulse, the apex beat and the influence of the heart rate on the irregularity. When possible the graphic method, such as the

electrocardiograph, should be used to verify the diagnosis.

Auricular fibrillation is distinguished from all other forms of irregularity by an entire absence of rhythm. It is extremely disorderly in character. The unequal spacing and force of the beats is best appreciated in taking the blood pressure.

As a general statement, it may be said that any irregular pulse of one hundred and twenty or more is almost always auricular fibrillation. Especially is this true if a cardiac insufficiency coexists. A slow heart with irregular action may sometimes be met with in auricular fibrillation, but it may also be due to the presence of extrasystoles or to a partial heart block. The exercise test or an electrocardiogram will make this clear.

Sometimes in a sinus arrhythmia there is a total irregularity but the rhythm is said to be regular as the rate is increased.

Flutter may present an apparently total irregularity although Lewis has shown that measurements of grouped beats, so-called spacing, show an element of regularity. With a rate over one hundred this and extra-systoles with partial heart block are practically the only conditions that simulate fibrillation.

It is important to remember that an increase of pulse-rate tends to abolish extra-systoles and sinus arrhythmias, whereas in fibrillation the irregularity is not abolished but is usually increased and the simulation of a dominant rhythm disappears.

In every case of cardiac arrhythmia the radial pulse and apex beat should be studied together. In auricular fibrillation there is a constant disproportion in the rates of the two, called the pulse deficit, as before described.

Where a mitral stenosis is present the presence of auricular fibrillation causes the rough crescendo presystolic murmur to be replaced by a long diastolic decrescendo murmur. Sometimes the presystolic murmur disappears entirely.

The prognosis of auricular fibrillation depends entirely on the integrity of the myocardium. The time of occurrence of the fibrillation is of some importance. An acute attack may occur and subsequently disappear, never to return, or it may be simply the first attack with many subsequent recurrences. It is the rule that it tends to recur many times, finally in long, severe cases becoming permanent. Expectancy of life can be based on two points, first, the response to treatment, and second, response to effort, that is, testing out by proper methods the heart muscle sufficiency before treatment has been instituted. How much unimpaired heart muscle is left possessing the ability to respond to proper medicinal and hygienic treatment? No safe opinion can be given until the patient has had the benefit of proper and prolonged treatment. Auricular fibrillation is no absolute indication of the condition of the heart muscle. A patient with a completely disordered rhythm may be able to perform the work and duties of his life without any definite evidence of beginning heart muscle failure. In such an event the prognosis would be favorable. On the other hand if he cannot perform the ordinary tests of life without signs and symptoms of cardiac impairment, the prognosis is relatively bad.

Even though the myocardium should be intact the onset of the irregularity is usually of serious import and may be a permanent disturbance of the auricle. In other words, the appearance of a fibrillation adds gravity to the prognosis if an organic heart lesion is present; failure being bound to eventually come. Auricular fibrillation, however, does not enable us to be any more accurate in our prognosis as to the duration of life and we should be careful in making absolutely bad prognosis. Lewis[19] states that "it loads an already defective muscle with an extra and appreciable burden. In most cases it heralds cardiac failure, temporary or terminal, so that few patients survive its onset for more than ten years." Osler[20] states "the condition may last for many years when once established . . . An unduly grave view must not, therefore, be taken unless there is marked evidence of cardiac failure." Mackenzie[21] adds "there are many individuals with fibrillation who lead useful and energetic lives, and whose capacity for work is little if at all impaired by the rhythm. In such the prognosis is distinctly good." Pardee[22] emphasized the necessity of continued treatment and concludes that the irregularity itself adds little or nothing to the gravity of the prognosis of the individual. Musser[23] reports a case of twenty years' duration and at no time did the heart muscle show evidence of an insufficiency. F. A. Willius analyzing the results in five hundred cases of fibrillation concludes that the mortality attending auricular fibrillation doubles and in some instances trebles that occurring in similar types of heart disease not complicated by this arrhythmia. Jones[24] in discussing the prognosis of a fibrillation has said that the senile

type is more serious than the rheumatic, that auricular fibrillation makes little difference so far as longevity is concerned in mitral stenosis, that this arrhythmia improves the prognosis in aortic regurgitation, that marked cardiac enlargement is an important handicap, that females do better than males, and that abnormal Q-R-S complex of the electrocardiogram is a serious sign.

In the treatment of auricular fibrillation quinidine should not supplant digitalis but at times be used as a supplement. In any case of fibrillation the question which presents itself is whether to give digitalis or quinidine.

The following criteria[25] for selection of cases most likely to be benefited by quinidine will be found useful:

1. A good myocardium, that is with little or no cardiac hypertrophy.

2. Little or no signs of heart failure, or in which the symptoms of heart failure clear up promptly after treatment is instituted.

3. Where fibrillation is of recent onset and especially if of short duration.

4. Where there is no serious valvular damage to the heart.

5. Not to be used in cases of thyrotoxic origin.

6. Not to be used in cases where the causative factor is of a somewhat similar active agent, as in rheumatism, in rare cases of malaria. (Eliminate first the causative factor.)

7. Can be used to an advantage in fibrillation following the poisons and infections.

White is of the opinion that any chronic fibrillation of one year or more standing is a contraindication to the use of quinidine, because:

1. Any return to normal rhythm is of short duration.

2. The danger of embolism, arising from a clot formed in the auricle during fibrillation, is increased.

3. Respiratory paralysis is more likely.

4. Sudden death has resulted, the cause of which is unknown, but possibly due to ventricular fibrillation.

Quinidine is a protoplasmic poison and acts by increasing the refractory period of the auricular muscle and decreases its irritability and conductivity. Its action is both upon the auricular muscle and through the vagus. In ordinary doses the heart is slowed and the auriculo-ventricular conduction time is lengthened. We should remember that the patient must be compensating with rest, morphia, digitalis, etc., before quinidine is ever given.

Untoward symptoms of quinidine administration are: headache, vertigo, nausea, vomiting, urticarial rash, convulsions, palpitation and faintness. In most cases the pulse increases in rapidity before the normal rhythm is restored. Toxic effects may appear after the establishment of a normal rhythm. The drug should be immediately stopped in any case where toxic symptoms appear. The onset of frequently extra-systoles or a persistently high ventricular rate affords an indication for cessation of therapy also.

The drug is rapidly eliminated and it appears that no cumulative effects take place. So far as it is known, the drug has no permanent effect. Approximately fifty per cent of the cases of flutter and fibrillation have been restored to a normal rhythm which persists from any number of days to years.

The sulphate of quinidine is given in the powdered form. Each gelatin capsule should contain five grains. Preliminary doses of five and ten grains are given on the first and second day of treatment, respectively, to test for idiosyncrasy. It is then increased to fifteen, twenty, thirty and forty grains daily until a change, if any, in the rhythm occurs. The daily amount should be given in divided doses. When the normal rhythm is restored this is gradually reduced until five grains is taken twice or three times a day. This should be kept up an indefinite period of time.

A series of fifty-three cases treated in the last five years has recently been reported. Thirty-eight, or seventy-one and seven-tenths percent, returned to sinus rhythm. Of these twenty-five, or forty-seven and one-tenth percent, remained regular for one month or more, fifteen, or twenty-eight and two-tenths percent kept their sinus rhythm for at least six months, three for at least three years and one is still regular after five years.[26]

In conclusion we should remember that the successful treatment of auricular fibrillation with the sulphate of quinidine depends on the proper observation and continuance of dosage even after the normal rhythm is restored.

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DISCUSSION

Dr. Caldwell, Lake City:

I was very much interested in Dr. Folsom's excellent paper, but was not aware until this moment that I was to be asked to discuss it.

Recently I saw a fresh specimen of a heart; the patient having had for a long time auricular fibrillation. This heart contained in one of the auricles a thrombus of about 4 cm. in diameter, and this clot had adhering to it a number of crumbling portions. These could easily separate off and float out in the blood stream.

I do believe that where we have an enlarged heart with long standing fibrillation it is dangerous to use quinidine.

From a diagnostic standpoint, I am very much interested in the electrocardiograph. In flutter we know that the auricular beat is pronounced in all leads in the electrocardiogram, but in fibrillation we frequently see many cycles in which there are no definite auricular beats visible. Again they appear nicely and again fade out. Just what happens I do not know. Perhaps, as Dr. Folsom says, "the heart trembling in diastole ceases to tremble rhythmically." White says of this matter, "it is almost certain that in auricular fibrillation there is a constant circus movement, but of course it is not a regular one or one that always follows a uniform path. Its irregular course and probable conflicts due to some areas of block account for the small irregular occilations in the electrocardiograms in auricular fibrillation."

At present I am treating one case of auricular fibrillation with digitalis and quinidine combined. I am glad Dr. Folsom mentioned that method in his paper. I used originally small doses of quinidine fourth day and night, never exceeding twenty-four grains in twenty-four hours, but recently, in one case, we gave as high as ninety grains in twenty-four hours before the rhythm became regular.

Dr. W. C. Blake, Tampa

(Note—This discussion was read by Dr. Spiers for Dr. Blake.)

We are greatly indebted to Dr. Folsom for the direct and clear-cut treatment of his subject.

That the condition he presents is frequent is evidenced by the fact that in about fifty percent of patients admitted to hospitals with advanced cardiac decompensation, auricular fibrillation is present. I know of nothing that is a greater test of one's diagnostic acumen than the ability to differentiate the cardiac arrhythmias. As has been said, "know auricular fibrillation and you know the arrhythmias." He who can sit by the bedside and correlate the heart rhythm, pulse rate and jugular pulse must be a keen observer. Mackenzie has given us one of the most graphic and inspiring examples of this painstaking and meticulous observation in modern medicine—the country practitioner who arrived at great truths by the careful correlation of the heart sounds and the visible or palpable pulsations of the circulatory apparatus. Not every case of arrhythmia can be differentiated without the electrocardiogram, but every effort should be made to arrive at a definite conclusion before the tracings are studied.

Auricular fibrillation almost invariably has as its background some profound degenerative cardiac lesion. While it at times occurs as a result of some acute intoxication, usually one attack is the forerunner of other attacks. It is therefore important after the attack has passed that a careful survey be made of the circulatory apparatus, especially the myocardial reserve. In this way the patient's activities and mode of living can frequently be so regulated as to prevent recurrence.

Quinidine sulphate is a decidedly helpful drug in both auricular flutter and fibrillation. It should be distinctly borne in mind, however, that it is an adjunct to, and not a substitute for, digitalis. In addition to depressing the vagus it acts directly on the heart muscle to slow conduction and to lengthen the refractory period. The contractible power of the heart muscle is at times so depressed as to increase decompensation. For this reason it should not be given in the face of severe decompensation.

Dr. M. J. Flipse, Miami:

I do not agree with one of the previous speakers, particularly as to the dosage he mentioned. There are a number of cases of auricular fibrillation that cannot tolerate large doses, and will get bad symptoms from them. There is a certain type of individual who will begin to show symptoms of intoxication when the dose is over 15 grs. It has also been my observation that the patient who is going to be benefited by quinidine will ordinarily be benefited following the primary large dose, the continued dosage should not exceed 6 to 10 grs. a day.

I am rising merely because I know there are some general practitioners in this meeting, and I would like to have you bear in mind the fact that quinidine is a dangerous drug. I do not believe that the toxic effect of quinidine on the heart muscle is the primary danger—but the indiscriminate use of quinidine in cases of auricular fibrillation of long duration. I do not think that they will receive any permanent benefit, and it is in these cases that we usually see the bad effects.

The opinion as to administering quinidine with or without digitalis: It will operate in both instances, but smaller doses of quinidine will operate if digitalis is not given, primarily because digitalis prolongs the interval between the auricular and ventricular contractions, and, in the prolongation of this interval, induces fibrillation in a person who is susceptible.

The more recent papers that I have read on this subject would advise the administration of quinidine every other day unless the dosage of quinidine which produces the effect is small. It not infrequently occurs that after the administration of a single 3 gr. tablet of quinidine, the patient who has been fibrillating for several days will stop fibrillating. That is the ideal type. But, when a patient comes in who has been fibrillating for three or four years it is, in my opinion, useless to give him quinidine.

I think if the general practitioner would commence the use of quinidine in 3 gr. dosage three or four times a day, as soon as fibrillation occurs, it would oftentimes re-establish a normal heart rate. The continued use when necessary of 6 to 10 grs. a day of quinidine might maintain rhythm —and we would then see fewer cases of fibrillation of long duration.

Dr. M. J. Myres, Daytona Beach:

I would like to add the following cause to the etiology of fibrillation mentioned in this interesting paper: While in charge of the Daytona Beach Boxing Club, I was greatly interested in examining the hearts of the men after a knockout. The effect of the knockout blow seems to bring on heart block, and this was followed by flutter and what seemed to be fibrillation.

INSOMNIA*

H. Mason Smith, M. D.
Tampa.

Any definition of sleep, like a definition of life, is inadequate. Our lives consist of the alternating waking and sleeping states and in this way the matter of defining one is analogous to defining the other. However, the characteristics of sleep are rythmicity, reversibility, suppression of responses to incoming stimuli and extinction of conscious activity associated with subconscious and somatic activity. Sleep is differentiated from stupor or coma by reversibility or wakefulness, from syncope or fainting by circulatory phenomena and evanescense.

Sleep is an avenue of escape from reality in some neuropathic individuals or a symbolic interpretation of the death wish; as in a case reported by Tucker[1] and it is probably on this characteristic that Washington Irving based his

*Read before the 57th Annual Meeting of the Florida Medical Association, Pensacola, May 6, 7, 1930.

story of Rip Van Winkle. Sleep as an instinct is analogous to the migration of the birds with the seasons or to hibernation in some lower animals.

Many theories have been advanced as to the cause of normal sleep; one was that the ganglionic cells of the brain retracted their dendrites which interrupted conduction and stopped the activity of the organ. This brought about sleep. Another interruption theory was advanced by Pukinje who thought that congestion in the basal ganglia blocked the corona radiata which run through them and brought about sleep. There were still other theories of this kind. It is not necessary in this discussion to do more than to state that all these theories of interruption are unsatisfactory and are of no value.

The idea of sleep as a conditioned reflex was advanced by Pavlov. This was the result of his experiments in conditioned reflexes on animals. The tendency of the animals to sleep as a response to conditioned stimuli was so great that his purpose was often defeated. A conditioned reflex is one excited by an indifferent stimulus through the mental association with the real stimulus; as the flow of gastric juices on hearing a bell which is associated with the giving of food.

Another group of theories as to the cause of sleep have their origin in the symptoms that are produced by sleep, such as cerebral anemia which does play an important part in producing sleep, but which does not explain the character of sleep and the periodicity of it.[2]

There have been numerous theories as to the cause of sleep which might be termed chemical theories, one of which is the asphyxiation of the brain due to carbon dioxide. Another is that the nerve cells have a changed appearance after exhaustion which is due to absorption of cell matter from them during the activities of the waking state.

The French physiologist, Pieron, took the blood serum of dogs that were over-exercised and had not been allowed to sleep for some time, and injected it into wakeful dogs which had had sufficient sleep. This produced prompt slumber on the injected dogs. Since this, it has been accepted that activity in the body during the waking state produces a fatigue substance as well as carbon dioxide which exercises a narcotic influence on the brain. This fatigue substance is eliminated during sleep. This theory also explains the periodicity of sleep, but cannot be the exclusive cause because we sometimes sleep without fatigue.

The endocrine theory of periodicity of sleep has been advanced and it is very plausible as it develops the idea of a balancing change of activity in two groups of glands.

The appearance in Europe of the epidemic of lethargic encephalitis which was described by Professor C. von Economo refuted all the foregoing theories and by working out the pathogenisis of this disease he has located and outlined an area of grey matter, the function of which is the regulation of sleep and in the broader sense of the word is a sleep center. Economo showed that in the somnolent-ophthalmoplegic form of this disease; that is a form with a somnolence which may range from a light sleep to a continuous soper in which patients may sleep for weeks, but in most cases can be aroused, associated with eye muscle disturbances as diplopia, ptosis, ophthalmoplegia, the pathology is an inflammation in the posterior portion of the lateral wall of the third ventricle which is adjoining the nucleus of motor oculi nerve.

In the other form of encephalitis which gave the initial symptoms of choreatic unrest with a tormenting insomnia the inflammation was found anteriorly in the lateral wall of the third ventricle near the corpus striatum, where, according to knowledge we already had, chorea-form diseases originate.[3]

As the inflammatory processes extend frontally or caudally symptoms may occur in accordance with the structures involved. There are many other disturbances of sleep besides somnolence and insomnia produced by encephalitis, such as the inversion of sleep, somnambulism, etc.

Now, there are a number of other diseases affecting this area which produce a similar symptomatology, namely, tumors of the infundibular region do this occasionally as an early symptom and all cerebral tumors cause soper as a late symptom, hemorrhagic areas in the walls of the third ventricle, Wernicke's and Gayet's disease.

Inasmuch as any condition or disease affecting this area causes the same reaction we consider that the proof is absolute that the function of this area of grey matter is the regulation of sleep and, therefore, it is termed the center for sleep regulation.

This sleep regulating center probably acts by coordinating the vegetative and psychic system to the changes which occur in sleep; that is changes

in respiration, circulation and bodily functions (demonstration of slide).

Now the practical value of this knowledge is twofold: first, as to localizing cerebral lesions; next, for the things that are to be hoped for in a therapeutic way, in the direct treatment of insomnia.

Already experiments have been made that justify us in believing that the time will come when we can influence sleep directly through this center. Hess of Zurich has succeeded in making cats fall asleep immediately with all the symptoms of normal sleep, by the induction of a very weak electrical current through fine electrodes to the posterior wall of the third ventrical and the anterior region of the aqueduct.[4]

Some results have also been obtained with diathermy. Probably this will some time be an agent that will relieve insomnia and it will not be necessary to prescribe the already overworked hydrotherapy, psychotherapy, and finally hypnotic drugs in the treatment of this annoying condition.

Sleep is the product of such a complex mechanism that a sleep center does not answer all the requirements. So it is a very reasonable assumption of many investigators that the fatigue substance in the blood sets this center into action.

The great Russian physiologist, Pavlov, made experiments on conditioned reflexes in animals which show that the prolonged or repeated action of a conditional stimulus leads to drowsiness; for instance, the flow of gastric juices in dogs is increased on hearing the dinner bell, which is associated with the idea of giving food, but when after hearing the bell repeatedly and no food is given a sleep is caused.[5]

Now it is known that prolonged action of some cortical areas leads to an inhibition of the neighboring cortical areas, and this inhibition spreads generally over the remainder of the cortex and causes sleep. Thus it is suggested that multiconditioned reflexes are a factor, probably the most important one in setting off action in the sleep regulating center.

Since insomnia is a symptom of physical disease or mental condition even though a severe symptom, probably the earliest or only symptom, the chief complaint that is brought by the patient, our recourse at present for the relief of this condition is to find the underlying cause and attack it.

As a matter of convenience and for clinical purposes with reference to therapeutic ends, we have made the following simple classifications:

1st. Insomnia due to physical disease.
   (a) Vascular.
   (b) Toxic.
2nd. Insomnia from external irritation, noise, climatic condition, etc.
3rd. Insomnia of mental origin.

The diagnosis and treatment of the first type is really a problem of general medicine. However, it is wise to remember that insomnia may be the prodromal symptom of an approaching disease and this complaint should lead to careful examination before it is placed in either of the other classes. In passing I may state that the vascular causes include both the high and the low tension types, both of which cause a cerebral hyperemia with which sleep is impossible. It also includes that distressing insomnia which is associated with chronic heart disease.

The toxic is the commonest form met with in practice and takes into its scope all toxemias from the remotest focus to the severest uremia and infectious processes.

In the second type, in those insomnias due to external irritations the treatment is obvious. But one comment should be made here.

A great number of neurotic patients attribute their insomnia to noises, especially if they should happen to be in a hospital, sanitarium or nursing home. They claim that they are in a noisy part of the building; that the noise is unbearable. It is usually safe to disregard this complaint of such patients to the extent of refusing to move them to another room. "One hears only the noises he listens for." And in these cases the sleeplessness is due to anxious expectancy.

The third group is that great class of people who are sleepless from psychic causes, which has inspired the writing of this paper as the experience of the writer has been greater with this class and the results of the treatment have been more varied than in the other. It should be realized that this group contains numerous types and that the particular mental causes are as numerous almost as the individual concerned, but they fall into certain general classes.

The most common psychological factor is overt worry or anxious preoccupation and this is found in the large number of people who have fallen into the habit of doing their thinking and planning after going to bed, outlining their next day's work,

and recapitulating all the events of the previous day, especially if some have not been profitable or pleasant.

Then there is another type of patient who denies that he worries when he goes to bed. He states that he goes to bed and "thinks of nothing." This is the man who worries all day and then expects to go to bed and change the form or the topic of his preoccupation. What he is actually doing is making a tremendous effort to repress his daytime perplexities, and this effort keeps him awake. The only treatment for this type of patient is removing his daytime anxiety.

There is another anxiety state which is secondary to both types just mentioned; that is anxiety which occurs over the loss of sleep. There is existing in the lay mind a profound impression of the disastrous results of the loss of sleep, so the fear of insanity follows and this fear or anxiety is then the most potent factor in producing wakefulness.

The writer recently observed a patient who lost so much sleep in recapitulating over his financial losses during the depression following the real estate boom that he began to visualize the most terrible possibilities of his plight. He was afraid nothing could now save him from insanity on account of the destruction of brain cells that had taken place during his sleepless nights which he had spent walking in the park. This fear was so tormenting he was reluctant to confide in the writer as a physician. After his confession and when he was assured that the loss of sleep had the most trivial effect on the mind or body and was never the cause of insanity, he immediately relaxed and dozed on the examination couch. After this fear was relieved his insomnia was cured.

The experimental data that have been obtained on the loss of sleep show that the effects in man are of a trivial nature. One can truly say that clinically the loss of sleep has never been an important factor in the causation of any mental illness.[6] The experiments at Colgate University on students revealed that the loss of sleep was attended with reduction of mental alertness, and emotional stability, but nothing of a destructive or irreparable nature.[7]

Any patient who suffers from insomnia, whether he expresses any anxiety or not over the disastrous effects of the loss of sleep, should be given the benefit of this comforting assurance as a therapeutic measure.

Probably the repression that causes most restlessness is that of the moral conflict. The statement of a person that he has not slept well usually brings the comment of a bad conscience. It is often a bad working conscious mechanism that causes it. The "would be" sleeper endeavors to keep his ethical failures pushed back in the lower strata of his unconscious and they are constantly struggling to get into consciousness and thereby causing a wakefulness.

There are many cases of insomnia which begin in response to some cause for anxiety and still persist after the original cause has disappeared. In these cases some inquiry should be made into the general attitude and emotional stability and usually a defect in the nervous make-up is found.

The methods of inducing sleep that are commonly employed are as a rule provocative of sleeplessness. The counting of sheep jumping the fence and other things which require mental effort produce a cerebral hyperemia which makes sleeping impossible.

In all the insomnias resulting from mental causes we see the conditioned reflexes spoken of above blocked. Any voluntary effort to produce sleep breaks these reflexes. We cannot possibly will ourselves to sleep. We allow ourselves to succumb to sleep which is brought about by these stimuli of the conditioned reflexes.

The usual sleeping time, the usual preparation for going to bed, such as undressing, the gradual recession of interest in the events of the day, the allowing the mind to wander at will without any conscious direction and finally the lying down in a comfortable bed and complete muscular relaxation are the stimuli that bring about the conscious inhibition and sleep. To go to bed in the middle of an enthusiastic discussion or argument without the relinquishment of mental activity is analogous to eating while on parade. Both acts are out of congress with the associated events and the former will not be followed by sleep and the latter will not be followed by digestion.

The writer is conscious that after every effort has been made to find the psychic causes producing insomnia and that one has done all humanly possible to eliminate these causes that in some patients insomnia still exists, and the patient goes on with this annoying discomfort. Is it too much to hope that since von Economo has localized the center regulating sleep and Hess has produced sleep by inducing electricity through this area,

that the time will come when we can relieve this distressing symptom by direct influence on the sleep center?

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DISCUSSION

Dr. W. H. Spiers, Orlando:

Dr. Smith has covered a very unusual subject. In fact, I have never heard a paper just exactly like it. Insomnia is a condition that has such varied symptoms and such varied causes. I will not discuss the physiological causes of sleep, and there is very little for me to say. But, insomnia is such a common malady and one of the most annoying conditions to be afflicted with. The causes are so many and varied that it takes quite a study of the individual to determine what to do for him. I am of the opinion, however, that in these cases there is bound to be some organic lesion. To some of the psychoanalysts there are causes that are cured by suggestion, but unlike the cases of the psychoanalysts, there is, I believe, in these cases an endocrine disturbance or some other source of toxins, such as mild infections.

This is a very hard paper to discuss, and a very hard paper to prepare, but I have received some suggestions from it that I believe I can utilize.

Dr. J. Q. Folmar, Chattahoochee:

I did not come over here expecting to discuss this paper; however, I have enjoyed it very much. I think the matter of the physiology of sleep is about as complicated as the physiology of some of the various autonomic systems of the body, for instance, that of respiration. We are definitely sure that there is a central respiratory system, and I believe that many of the studies made with reference to central encephalitis and some intracranial organic lesions located in the various sections of the brain lead us to believe that there is equally a central sleep center.

This is a subject that to me is very interesting, but the more I study it, the more confused I become with reference to the physiology.

Dr. Smith, Tampa, (concluding):

I have nothing further to add.

ARTIFICIAL PNEUMOTHORAX IN THE TREATMENT OF PULMONARY TUBERCULOSIS*

M. Jay Flipse, M. D.
Miami.

The intentional collapse of a lung for the treatment of pulmonary disease is a comparatively recent procedure. First recommended on theoretical grounds in 1821 by James Carson of Liverpool, it was not until the beginning of the present century that this method of treatment found application in more than a handful of cases. James Houghton in 1832, and Stokes in 1837, pointed out the not infrequent beneficent influence of spontaneous pneumothorax in certain cases of advanced phthisis. Credit for the first pneumothorax treatment is given to Potain who in 1884 injected sterilized air to replace fluid in a case of spontaneous hydro pneumothorax. During the following five years he treated two other cases by the same method, reporting all three in 1888. At about the same time, in 1885, Cayley treated a case of severe tuberculous hemoptysis by open incision of the chest wall, reporting the case to the Clinical Society of London.

During the next ten years, Forlanini treated several cases in a similar manner, reporting them in 1894 and 1895. Three years later, J. B. Murphy of Chicago reported five cases in which pneumothorax was induced by trocar and canula. In 1899, Schell of Indiana used pneumothorax to control a case of hemoptysis, and two years later Lemke reported his results with artificial pneumothorax in 53 cases of pulmonary tuberculosis. From 1905 to 1910, a number of cases were reported by Brauer, Schmidt, Forlanini, Dumarest, Saugman and others. The pneumothorax treatment thus gained rapid recognition in all countries. Favored by practically all who had opportunity to observe its beneficial effects, it is now universally recognized as the one hopeful method of treatment in advanced tuberculosis.

In bringing it to your attention, it is not our motive to offer any contribution of original character. It is rather in the hope that you may become familiar with the indications and contra-indications of pneumothorax together with the results which can be obtained in cases otherwise hopeless. We have availed ourselves liberally of the useful material which has come to our atten-

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tion, and extend appreciative acknowledgment to the sources indicated in the appended bibliography.

Indications for Artificial Pneumothorax

1. Chronic cases of pulmonary tuberculosis in which the disease is clinically unilateral, and in which the usual methods of treatment have been unsuccessful.

2. Cases of unilateral distribution where, for economic reasons, patients are unable to spend an adequate period of time under the usual rest regime and in whom toxemia prevents moderate activity.

3. Unilateral activity with cavitation.

4. Tuberculous hemoptysis where life is threatened by loss of blood, and where the source of hemorrhage can be definitely determined. Here pneumothorax is indicated even with a fairly extensive process of the contralateral lung.

5. Certain cases of bilateral tuberculosis which have failed to improve under ordinary measures of rest in bed.

Contraindications

In general, extensive disease of various organs, whether of tuberculous etiology or otherwise, precludes artificial pneumothorax.

1. *Lungs:* Extensive bilateral tuberculosis involvement as a general rule contraindicates artificial pneumothorax. However, even in a few selected cases of this type, a partial collapse has occasionally been of benefit. There is a growing tendency to try partial pneumothorax, even bilateral partial collapse in cases which otherwise must terminate fatally.

Advanced emphysema and asthma are definite contraindications. A cautious trial of pneumothorax is, however, justified even in these cases unless dyspnea and cyanosis are present.

2. *Heart:* Serious heart disease, especially of acute nature, does not tolerate pneumothorax. Because of the derangement of pulmonary capillary circulation with a reduced vascular area after collapse of one lung, the right heart must be in fairly good condition to tolerate this procedure. Yet it is often surprising what excellent results are sometimes obtained in spite of chronic cardiac conditions such as myocarditis and arteriosclerosis.

3. *Kidneys:* Acute or chronic nephritis of a severe grade would seem to be a definite contraindication. It must be borne in mind, however, that sometimes an apparent nephritis may be an expression of the tuberculous toxemia. Such cases usually improve after the reduction of absorption from the infected lung.

4. *Intestinal Tuberculosis:* It has been well demonstrated that advanced cases of this nature rarely improve after pneumothorax. Gravesen, Brauer, Spengler and Forlanini report that milder cases often show a decidedly favorable response when the auto-inoculation is removed by collapse of the infected lung.

There are, of course, other contra-indications which may appear in individual cases. Each case must be decided on its own merits after as careful a study as time permits. As experience with the method and with the results of ordinary measures of bed rest permit, the development of that peculiarly personal attribute of the physician, medical judgment, will gradually replace the "rule of thumb" decision, and determine without conscious mental processes which cases are suitable for collapse therapy, and which should be rejected.

Selection of Cases

From previous statements, it would appear that there is a rather wide latitude in the application of artificial pneumothorax. Actually in the study of any given group of cases, the number suitable for pneumothorax treatment will be found to be rather small. In a total of nearly 2,000 cases, four prominent workers in this field found from 3½% to 10%, or an average of 7%, immediately suitable. It must be borne in mind, however, that nearly all patients at one time or another during the course of their disease have been, or will be, candidates for artificial pneumothorax. Therefore, the responsibility of the physician is increased in that he must not only discover tuberculosis early but bear in mind the possibility of benefiting the patient by a timely collapse if the patient does not improve under the ordinary measures of bed rest.

Palliative Pneumothorax

In certain quite hopeless cases, Saugman reports great temporary improvement from partial collapse. Pisani and Forlanini also feel that even in extensive cases where there is "everything to gain and nothing to lose", the patient should be given the chance with pneumothorax. In these cases, the method differs considerably and instead

of a complete collapse, only a partial collapse is desired. In effect, a partial collapse reduces the toxemia, and often benefits the contralateral lung by relaxation of the mediastinum. Cough, sputum, temperature and aspirated caseous bronchopneumonia are often reduced. These cases are always bilateral and the chief consideration is the progress of the contralateral lung. It has been shown that quiescent or fibriotic apical lesions of the contralateral lung tolerate pneumothorax well; peribronchial and basal aspirated caseous involvement of the contralateral lung usually are aggravated by collapse of the other more advanced lung.

Failures in pneumothorax treatment are chiefly due to adhesions which prevent complete collapse, and in some cases prevent even a partial pneumothorax. In these cases surgical measures such as phrenectomy of thorocoplasty may still be used. Our present discussion, however, is limited to pneumothorax, so even though other surgical procedures are of marked value in selected cases, we will but mention them here without defining their application.

The percentage of failures in the induction of satisfactory pneumothorax depends upon the type of cases being treated. In the hands of various workers, it varies from 20% to 50%. The presence of minor adhesions does not interfere with pneumothorax and, in fact, is encountered in practically every case. It is in cases of obliterated pleural space with massive adhesions that pneumothorax becomes impossible. Since no method other than trial can determine the success or failure of pneumothorax, numerous cases are attempted where the tuberculous process is very extensive. It is surprising how frequently it is possible to induce at least a valuable, if not a complete collapse in the face of what at first appears to be an impossible situation.

Dangers of Pneumothorax

Like all surgical procedures, the induction of pneumothorax involves certain potential dangers which must be borne in mind. We will not emphasize these too strongly since by general concensus of opinion, the danger of not producing pneumothorax in a case requiring it, is far greater than the occasional serious or fatal result attributable to the procedure.

Included among the dangers are:

1. Pleural shock.
2. Air embolus.
3. Rupture of the lung.
4. Perforation of the lung.

Other possibilities are:

1. Puncture of diaphragm or pericardium.
2. Subcutaneous emphysema.

Fortunately, these dangers are minimized by certain precautions, and are very rare in the practice of experienced operators. Nevertheless, they exist not only at the induction of the treatment but even at refill treatments. We do not believe that the frequency of these accidents is any greater than the occasional fatality resulting from the use of general anesthesia in other surgical procedures.

Complications of Artificial Pneumothorax

By far, the most frequent complication of artificial pneumothorax is pleurisy with effusion occurring, according to Riviere, in about 50% of the cases. The quantity of fluid varies, and is not especially important except that re-expansion of such a lung is usually difficult or impossible after hydro- or pyo-pneumothorax. The fluid is usually sterile, though temperature may be present due to tuberculin sensitization. After a time, the fluid is absorbed, only occasionally need it be aspirated.

Extension of tuberculosis in the contralateral lung can only questionably be considered as a complication due to pneumothorax. There is a greater probability that such extension occurs in spite rather than because of collapse of the other lung. Nevertheless, if such extension does take place, it at once raises the question of the advisability of discontinuing the pneumothorax treatment.

Incidental colds and pneumonia of the functioning of the lung constitute one of the gravest accidents during pneumothorax. While not in any sense a complication due to the procedure, the seriousness of these infections is much greater because of the presence of a pneumothorax. Pneumonia usually terminates fatally. Fortunately in this climate, we have very little pneumonia. In seven years, I have seen but one case of pneumococcus pneumonia in the tuberculosis ward at the Jackson Memorial Hospital at Miami. This patient was not under pneumothorax treatment, but had a spontaneous pneumothorax which had induced about 33% collapse of one lung. He also had bilateral cavitation. In spite of this, he failed to fulfill our prognostication and after a stormy course, the pneumonia terminated favorably with apparently no immediate exacerbation of the bilateral tuberculous infection.

Method and Duration of Pneumothorax Treatment

We have intentionally omitted a discussion of the method of giving pneumothorax treatment, because we did not wish to fatigue you with a theoretical discussion of a procedure which probably but few of you will ever use. Anyone who is especially interested can find an excellent description of the procedure in Riviere's monograph on this subject, or in other texts on surgical treatment of pulmonary tuberculosis.

We will devote a few minutes to the discussion of the rationale of the treatment. The purpose of pneumothorax is to permit a diseased lung to collapse under its own elastic recoil and possibly to compress it if necessary so that there is no longer any expansion under respiratory movements of the chest. With the lung in a condition of full collapse, the blood supply is reduced, cavities are obliterated, and fibrosis takes place to a varying degree in different cases. After a period of time adequate to allow healing, it is the hope of the operator that the healthy parts of the lung will re-expand, and permit a certain degree of function, but that the diseased part of the lung will be firmly fibrosed.

Collapse is induced by introducing a gas into the pleural cavity under controlled pressures, and in measured quantities. Since any gas is gradually absorbed, it must be replaced at intervals to maintain the degree of collapse and compression required for the individual case. In practice, air is the gas used most frequently. The collapse is induced gradually and refills, which at first are required every few days, eventually need be given only at intervals of once in two to eight weeks. To be effective, collapse must be maintained from one to five years. Except at first, the ordinary cases are ambulatory, and frequently able to work, thus becoming economically independent during the course of the greater part of the treatment.

Results of Pneumothorax

1. Reduction of the toxemia. It is often little short of miraculous how patients with fever, cough, night sweats, loss of appetite, and all other evidences of a severe infection will, after pneumothorax treatment, lose all these symptoms and be able to go about their business in a fairly normal manner.

2. Limitation of spread of the disease. It is the observation of all workers in this field that the rapid advance of pulmonary tuberculosis is usually due to aspiration of infected sputum with the production of caseous bronchopneumonia. A collapsed lung produces no sputum, and the danger of spread by this method is markedly reduced. The blood and lymph supply is also markedly reduced so that miliary tuberculosis is less frequent.

3. Reduction of danger of hemorrhage.

4. Reduction of infectiousness. The sputum becomes free of tubercle bacilli in about 50% of far advanced cases with pneumothorax, thus reducing the danger of the spread of the disease to others. This is of tremendous economic importance and will doubtless be reflected in the statistics during the next decade.

5. Prolongation of life.

While a proper length of time has not yet elapsed since this method of treatment has come into general use, it is possible to draw some conclusions from the results of earlier workers which point definitely to pneumothorax as a valuable therapeutic measure. When it is recalled that most of the cases treated are third stage—far advanced, sputum positive—the statistics are even more remarkable.

Saugman reports in a series of 310 cases completed, that at the end of ten years, 30% of his series were fit for work. Comparing these cases with those in which pneumothorax could not be accomplished because of adhesions, he found none in the later group fit for work after the 8th year. Bonzoni collected statistics of 3,680 cases treated between 1916 and 1925, including 71 under his own observation. Of the total, 25% were described as cured, 27.1% improved, and 31.29% dead.

The Matsons and Bisaillon have indicated that three factors enter into the end results of pneumothorax. They are in order of their importance:

1. Type of disease.
2. Character of pneumothorax.
3. Status of the contralateral lung.

Regarding the type of the disease, the fibrocaseous offer the best results with 40% clinical recoveries as against 23% to 27% recoveries in cases with cavitation of different degree.

Regarding the character of pneumothorax, Gravesen's figures published by Saugman showed at the end of 3 to 13 years, 70% of the

cases of complete pneumothorax were fit for work and 22% were dead. In those in whom partial collapse only was obtained, but 11% were fit for work, and 58% were dead. Cases with no free pleural space showed 5% fit for work and 66% dead.

The condition of the contralateral lung has been shown by the Matsons to be of less importance than the other two factors. In a series of 282 cases with demonstrable disease in the contralateral lung in only 34 cases was there any extension of the disease, and of these, 22 died. These constituted only 16% of the fatalities in their series of 480 cases. Apical fibrocaseous disease of the contralateral lung of active or quiescent character gave 48% and 50% recoveries against 52% in cases with no disease of the contralateral lung. Aspirated bronchopneumonia of the contralateral lung gave poorest results with only 26% recoveries.

The important point in these statistics is that best results are obtained only if pneumothorax is instituted promptly, and early in cases which do not give promise of complete recovery by simple hygienic measures.

DISCUSSION

Dr. W. A. Claxton, Melbourne:

I am sure we have all enjoyed this paper very much. It is very instructive, especially since we have not had very many papers along this line. The treatment of tuberculosis has gone through a regular cycle in the past number of years. Those of us who are familiar with the history of tuberculosis will remember that years ago they put a man on horseback even where the man was so weak he had to be held on the horse and made him travel around the country on horseback to cure tuberculosis. Later that method was modified a little bit to the extent of telling the man to get an outdoor job. Still later the question of rest became of paramount importance, and rest along with food and good hygienic surroundings was thought to be the best treatment for tuberculosis. And in most sanitariums rest is the principal factor in the treatment now. However, within the last few years the treatment of tuberculosis has changed from what we might designate as passive treatment to active treatment. Rest is still the greatest factor in effecting a cure but instead of allowing the patient to lie in bed for months or years and perhaps just holding his own or improving a little bit and having set-backs, the tendency now is to take up more active measures of treatment, and that is where the question of pneumothorax comes in.

With reference to pneumothorax, it is one of three surgical measures, each of which has been tried with good results, but pneumothorax is the ideal of these three methods. Thoracotomy is the first choice when pneumothorax cannot be utilized, and thoracoplasty is the third choice. Pneumothorax is the least harmful of these methods and in the majority of cases is very successful. I was connected with an institution once where we had about 250 patients and of these forty were taking pneumothorax. In another institution we had about 20 pneumothoraces out of 200 patients, so you can see the method is being used extensively. Our plan usually was to wait three months, and if the patient was not improving as he should we tried to establish pneumothorax.

I remember one patient who came in with much toxemia. The question of pneumothorax immediately came up. It was at that time a unilateral case. We waited about two weeks, then made up our minds to do a pneumothorax, but decided to take another X-ray picture. Much to our astonishment the disease had extended to the other side and was stationary in the original lung. We established a pneumothorax in the newly affected lung and by keeping the man flat on his back for eight months with pneumothorax we were able to get him in condition where he could get out of bed and go back to work. That was rather an outstanding case because the disease was progressing so rapidly.

In giving the details of this treatment there are one or two litle things that Dr. Flipse purposely did not mention: All pneumothorax refills should be controlled by X-ray. A fluoroscopic examination should be made of the patient before he goes in for refill and after he comes out. You can detect very small amounts of fluids by the fluoroscope where you possibly could not by physical examination and also can more carefully regulate the amount of air injected.

Dr. M. J. Flipse, Miami, (concluding):

I have nothing further to add.

SPINAL ANESTHESIA*

JOHN R. BOLING, M.D., F.A.C.S.,
Tampa.

My only excuse for presenting a paper on this subject is because of considerable personal interest in it, and the wide-spread attention that is being given it throughout the medical world. My own experience, although dating back some nine years, has been rather limited, and I present this more as a preliminary report.

I am not a fanatic on the subject. I do not think it is superior to all other forms of anesthesia as a routine, and I use ether in far more cases than the spinal. But it does have a very important place in surgery and there is a definite field for it. There are cases in which no other form of anesthesia can be used with anything like the safety that this can.

Especially since the war, greater progress has been made in anesthetics than in any other branch of surgery. Ethelyn has been put in use as a new agent. The old anesthesias and methods have been improved, and local anesthetics have been greatly advanced, being far less toxic than before.

Standing out boldly above the rest, and demanding a place in surgery, is spinal anesthesia. The day has arrived when any man practicing surgery or anesthesia must familiarize himself with this type of anesthesia. The method is firmly founded. It is accurate, scientific, and safe. During the past two years there has been much progress made, and the literature is filled with it, but it is not new. In 1899, Matas operated successfully with it; Babcock took it up as routine in 1904; Babcock and Boyd each have operated over twenty thousand cases with it.

The profession, until very recent years, has been under the impression that spinal anesthesia was to be used only in bad risks, in which it was unsafe to use ether. Naturally, if used in risks of this type only, it was inevitable that it fall into disrepute.

As we knew spinal anesthesia in the past, it had many features that were undesirable, and was considered unsafe for routine use. The duration of anesthesia was uncertain, intensity variable, failures frequent, nausea and vomiting troublesome. Post-anesthetic conditions, such as violent headache, paralysis of the sphincters, diplopia, temporary blindness, vertigo, and palsies, were not rare. At the time of anesthesia there was marked, often alarming, drop in blood pressure, the patient often being pulseless, and as Hillsman expressed it: "the blood pressure was nothing over zero."

One of the great fears experienced in the use of spinal anesthesia is that of respiratory paralysis as a result of the action of the drug on the medullary centers. The idea conveyed in most instances is that because of diffusion, the drug ascends in the spinal canal from the point of its injection, and if it reaches the medulla, depression of the respiratory center takes place to such an extent that paralysis ensues.

Koster and Kasman of Brooklyn, New York, have done a great deal of experimental work to discredit this apparent danger. They find that it is advisable to induce (rather than guard against) diffusion to the medulla and brain stem, in order to secure anesthesia of the entire body. If, as some claim, diffusion from the lower portion of the spinal sub-arachnoid space is ordinarily so slight that the amount of anesthetic agent which reaches the medullary portion of the cerebro-spinal system is insufficient to cause enough depression to result in respiratory paralysis, and that only occasionally does the concentration of anesthetic become great enough to induce respiratory depression and paralysis, then by direct application of a concentrated solution one should always be able to induce such respiratory depression and paralysis. This Koster and Kasman disproved by the direct application of neocaine solution to the medulla of both frogs and cats. In no instance, although there was a complete anesthesia with paralysis, was there any appreciable change in the respiratory movements of the animals.

Continuing to quote from their article, it is quite apparent that the anesthetic agent had a selective affinity for sensory nerve tissue. This being true, it is not difficult to understand how an anesthetic solution which diffuses up to the medulla or to the level of the region of the phrenic nerve, namely the third, fourth, and fifth cervical roots, could cause abolition of sensory conduction without any effect whatsoever upon the motive power of the respiratory organs.

Their clinical application of these experiments has proved, certainly to their satisfaction, that

*Read before the Hillsboro County Medical Society, Tampa, March 18, 1930.

one can obtain a complete anesthesia by spinal injection without fear of respiratory paralysis.

Personally, probably through lack of experience with this anesthetic, I prefer the anesthesia not to be complete. I have not up to this time operated above the diaphragm. Possibly, or rather probably, as time goes on, and my experience increases, I will fear it less. Certainly this has been true in the past.

This anesthetic prevents operative shock by blockage of shock impulses coming from the operative field and producing the "anociassociation" of Crile. It is indicated in advanced cardiac diseases, especially those of failing compensation, as the fall in pressure rests the heart and the burden is considerably lightened. It is of primary importance in pulmonary diseases and in late renal diseases; in hypertension, anemia, in diabetes, and in osteomyelitis.

The fall in pressure which sometimes occurs is not one of shock. It is due to anesthesia of the greater splanchnic nerve, supplying all the blood vessels of the abdominal viscera. As a result, there is a great dilatation of the splanchnic vessels, with a corresponding lessening of blood in the peripheral vessels.

Nine years ago, I made a trip to Philadelphia to see Babcock use this form of anesthesia. I returned with considerable of the enthusiasm that he had. Over a period of a year or more, I used it, but the majority of the cases were poor risks, more especially those of hypertension type. It did not take my enthusiasm long to wane, because of the alarming symptoms that were so prone to develop. Frequently the patients became pale, clammy, and pulseless, the pressure dropping over 100 mm. in some instances. I never had a fatality, but discontinued its use because of the fear of one. My experience was no different from that of others, and it was only the more courageous who continued in its use, and by steady experimentation have eliminated most of the ill effects, and given us an anesthetic that is safe.

The ideal spinal anesthetic would be one that would be certain in its effect, constant in duration, in direct ratio to the amount of anesthetic used; one that would not affect heart action, arterial pressure or respiratory function; one that would lessen nausea, pallor, and cold sweats.

George P. Pitkin, of New Jersey, has devoted a great deal of study to the solution of the many problems mentioned above, and has done much toward the elimination of danger. His technique and solution are gaining universal notice and use. He has prepared a solution that is lighter than spinal fluid, and the level of anesthesia is governed by the angle of the patient's position. Before the injection of the anesthetic into the spine, he injects the skin of the back with 1 c.c. solution, 1% novocain and 5% ephedrin hydrochloride, which prevents the blood pressure drop In fact, the usual thing is that there is an increase of five to twenty mm. in pressure for the first fifteen or twenty minutes, then a drop to normal, where it usually remains.

There has been much improvement in the purity of drugs in recent years, which has contributed much to the safety of spinal anesthesia.

Recently, we have been using this anesthesia oftener, and find it far superior to that of our previous experience. There has been a complete absence of the alarming symptoms mentioned. In none of these cases, with possibly one exception, has there been any sign of toxemia or shock. With the exception of the few in which the anesthesia has not been complete, the method has given perfect results. I am, therefore, viewing it very differently now than when I first used it.

There are many things in its favor over other forms of anesthesia. The discomfort of ether inhalation, which is such a fear to many, is thus eliminated.

We have, during our series of cases, used both spinocain and novocain, using from 75 to 200 mg. of novocain crystals for an injection, and spinocain from 1½ to 4½ c.c., each 2 c.c. ampoule of spinocain containing 200 mg. of novocain, and put up in a solution lighter than spinal fluid.

Pitkin claims for the spinocain that it is easier to localize the anesthesia by what he calls his "controllable anesthesia." I find it advantageous to use the novocain crystals, dissolved in spinal fluid in those cases where accurate angulation of the patient is difficult to obtain, or where the patient is to be injected in the bed before removal to the operating room. For example, in painful fractures of the lower extremity, it may be advisable to anesthetize the patient before moving, so as to eliminate this pain; or in the removal of a cast from a painful fracture of the leg that is to be operated, we inject the anesthetic

while the patient is still in bed, and the cast is removed before going to the operating room, obviating this tedious and dusty process in the operating room. Otherwise, we are somewhat more inclined to use the Pitkin solution of spinocain. We have gradually increased our doses as we lose our fear of the drug.

Romberger in a recent article states that Pitkin's technique of 2 c.c. of spinocain expanded to 8 c.c. does not give satisfactory anesthesia in the upper abdomen. He uses as routine 4 c.c. diluted to 8 c.c. for the upper abdominal and long cases, and should this not give satisfactory anesthesia, he re-injects.

In one case of a woman weighing 140 pounds, operation being cholecystectomy and appendectomy, he injected at the first dose 5 c.c. of spinocain expanded to 10 c.c. by spinal fluid. Ten minutes later, realizing the anesthesia was incomplete, he injected 3 c.c. of spinocain expanded to 6 c.c. After waiting another ten minutes, he gave a third injection of 4 c.c. of spinocain, unexpanded, and got a satisfactory anesthesia with no operative or post-operative concern, sensation and motion returning in one hour and thirty minutes.

Our preparation for spinal is practically the same as for ether, except we never give enema the morning of operation. This is because there is a paralysis of anal sphincter and lower colon, and of the inhibitory nerves to the entire gut. The motor nerve (vagus) not affected. This condition, of course, gives increased activity to peristalsis of gut. There is, therefore, the possibility of an involuntary stool which might soil operative field because of Trendelenburg position. It is also because of this that the gut is so easy to handle in abdominal cases. It is small because of the contraction. We give 1/8 to 1/6 gr. of morphine 1½ hours before operation and 1/8 gr. morphine and 1/150 atropine one-half hour before operation. When using spinocain for abdominal work, we inject 3 to 4 c.c. of the spinocain in the second lumbar space, after diluting it to 6 or 8 c.c. with spinal fluid. The injection is made through a small needle of a 20 gauge, with bevel of 45 degrees. This is always given with the patient on the side, never while sitting. Patient is then turned on back, with table level, or with head lowered a little. The anesthesia is watched, and when rib margin is reached Trendelenburg is increased to about 5 to 10 degrees. If anesthesia should not reach high enough then, 1 to 1½ c.c. spinocain is injected in the first space. For bladder work, or operation on lower extremities. 2 c.c. in the third space is sufficient.

When using novocain crystals, we use for abdominal work 100 to 200 mg. diluted to 4 to 6 c.c. with spinal fluid, in the second lumbar space. This can be given while sitting, because it is of the same specific gravity as spinal fluid. However, I prefer to give mine with patient on side. The level of anesthesia in this is not governed by the position of the patient as in spinocain. However, in all spinal work (with possible exception of Pitkin's heavy solution), the patient must be in Trendelenburg position to prevent anemia of the brain. Whether we give spinocain or crystals, we use the same preliminary injection of 1 to 2 c.c. of 1% novocain, 5% ephedrin solution.

Any danger that may come from spinal anesthesia lies in the anemia that may develop in the brain, and this is overcome by the Trendelenburg position. I believe it is unwise to do any operation under spinal in which it is not possible or convenient to put the patient in the Trendelenburg. In so doing, the cerebral anemia is overcome by gravity.

We have used spinal anesthesia in only a comparatively few cases, approximately ?? cases in both the first and last group. This is not many, but still sufficient to get a pretty good idea as to the value of the procedure. All of the latter group were within the past fourteen months. During the seven-year interval during which I did not use this anesthesia, there has been much improvement, and all my old fears have been gradually eliminated during the past twelve months, and it is with an increasing feeling of safety that we use it. We have done the usual operations below the diaphragm, including gall bladder, hysterectomy, intestinal obstruction, hernias, appendices, prostatectomy, fractures of leg, both of thigh and lower leg, debridments, etc.

The vast majority of these cases were given spinal because ether was contraindicated to some degree, for one reason or another, or by request of patient. The anesthesia in all but three was entirely satisfactory. One of these was last June. 2 c.c. of spinocain was given in the third space, followed by 1½ c.c. in the second space. Anesthesia was not complete and ether was

given. She simply did not have enough anesthetic. Within the past month, I have had to reinject two cases after having put 3½ c.c. in the second space, to go above to the first space and inject 1 c.c. more, and obtained perfect anesthesia in both. The day after I moved to Tampa, September 18th, I injected 2½ c.c. in the second space for hysterectomy, tested out skin and after twelve minutes, thinking I had anesthesia, began operating. Much to my chagrin, patient complained of pain. Gas ether was given for ten minutes, at end of which time I noticed the marked abdominal relaxation that is obtained only with spinal anesthesia. The gas ether was discontinued. The anesthetic was complete and the operation proceeded under the spinal. My mistake was in beginning before I was certain anesthesia was present. The other unsatisfactory case was hysterectomy for bleeding fibroid in an obese anemic woman. 3½ c.c. spinocain was given in the second space. The anesthetic was perfect, but patient complained greatly of nausea and retching, which was troublesome during operation. She had had morphine grs, 1/6, one and one-half hours before operation, and morphine 1/8 and atropine 1/150 one-half hour before operation. The nausea and retching continued until the following day when all morphine was discontinued. The nausea promptly subsided. I think the morphine was more to blame than the spinocain.

Other than these three, the anesthesia has been in every way satisfactory, and certainly in the first two the fault was mine, not the fault of the anesthetic agent.

There has been a marked absence of vomiting, abdominal distention and post-operative discomfort associated with ether. There is less shock, frequently the pulse on return to room being as low as when going to operating room. Without exception, the patients are delighted with the anesthesia. As I gain experience with it, my regard for it increases. The beautiful relaxation, which is such a great aid to adequate exposure, is seen in no other form of anesthesia.

In conclusion, let me say that the use of spinal anesthesia is certain to give one a high regard for it, and the eradication of the fear of it which most men now have. It is swift; it is, with proper technique, practically certain; it is controllable; and it is safe.

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CAUDAL ANESTHESIA IN RECTAL SURGERY

C. Larimore Perry, M.D.,
Miami.

Caudal anesthesia is a state of anesthesia produced by the injection of an anesthetic agent into the caudal canal of the sacrum, thus interrupting or blocking the sacral nerves which supply the rectum.

The procedure has become a valuable aid to the general surgeon who has been willing to acquire the simple technic necessary in the successful administration of novocaine in this manner. Much has been written, giving every detail in technic, and I feel that many surgeons have been discouraged in the use of caudal block because of the difficulties that may be met in following the technic as outlined in most text-books on anesthesia. It is with this in mind that I wish to present a technic that will get results and still not be a laborious procedure to follow.

The equipment necessary to produce caudal anesthesia consists of a fresh solution of one per cent novocaine, one ampoule of adrenalin, an ordinary luer syringe of ten cubic centimeter capacity, a spinal puncture needle that will fit the syringe, and a small hypodermic needle.

The patient is placed in a prone position on the table, with a pillow or sand bag under the spines of the ilium. The skin over the sacrum from the lower lumbar vertebra to the coccyx is prepared by first applying ether, followed by alcohol and iodine.

The two cornua of the sacrum are palpated at the base of the coccyx. The small hypodermic needle is attached to the syringe and a cutaneous wheal is raised between the cornua. With the same needle the sacrococcygeal ligament is injected with several cubic centimeters of novocaine. This injection should be followed by a brief pause, at which time the patient can be assured that he will not be subjected to further pain. The hypodermic needle is removed and the spinal puncture needle with the stilet in place is inserted through the wheal site and the sacrococcygeal ligament until bone is touched. The

butt of the spinal needle is then depressed and the point of the needle forced forward an inch. It is unnecessary to force the needle its whole length into the caudal canal, as the novocaine will permeate the loose fatty tissue if only its point is in the canal. Evidence that the needle is in the caudal canal consists of a free flow of novocaine from the syringe to the canal with very little pressure.

Forty to fifty cubic centimeters of a one per cent solution of novocaine is injected into the caudal canal slowly and the needle is then removed. Anesthesia is complete for rectal surgery in about fifteen to twenty minutes, and no effort should be made to proceed until this time has elapsed.

I have not found it necessary to resort to injections of the sacral foramen in performing operations on the rectum. It is my belief that most surgeons insist upon the parasacral injections to enable them to proceed without the twenty-minute period of grace. Therefore, caudal anesthesia without the parasacral injections is not meant for the surgeon who is impatient.

I have heard rectal surgeons say that the relaxation from the caudal injections is not complete, but when supplanted by parasacral injections the relaxation is complete. It has been my privilege to discount this contention by raising the cutaneous wheals along the sacrum so that the surgeon could see them, but not injecting the sacral foramen. The relaxation was suitable if the wheals were present.

Labat, in his excellent book on anesthesia, says that a show of blood from the caudal needle is evidence that the needle is properly placed. While this is true, I feel that the show of blood carries some moment in the production of novocaine reactions. The show of blood is not encountered if only the point of the needle is inserted in the caudal canal. The forcing of the needle along the bone will injure the venous plexus that lies in the caudal canal, and will open up the blood channels for a more rapid absorption of the anesthetic agent. I feel that this injury to the venous plexus is the cause of most of the reactions seen in this form of anesthesia. During the past six months I have made every effort to keep the point of the needle out of the venous plexus by not forcing it over an inch into the caudal canal. In a high percentage of cases this can be accomplished if the operator exercises care. Once the needle is inserted and there is no flow of blood from its lumen, it is presumptive evidence that the venous plexus has not been injured, and I feel safe in injecting the solution of novocaine with no concern of an impending reaction.

An alternative presents itself in those cases in which, with this technic, the sacral venous plexus is injured. The needle is withdrawn and the second sacral foramen on each side is injected. If by any chance this insertion of the needle hits a vein, the needle is withdrawn and the third foramen is injected. In other words, no solution is injected into a foramen in which the needle has injured a vein. At times this has been awkward, as the second foramen on one side may have been injected, and on the other side I am forced to rely on the third or fourth foramen before I reach a bloodless spot. The anesthesia will be the same regardless of the foramen used, provided sufficient time is allowed the solution to permeate the nerve trunks.

In following this procedure for caudal anesthesia I have had no novocaine reactions, and it is largely from the results of this procedure that I venture to assert that most of the reactions encountered are the result of venous absorption of a warm solution of the drug.

The practical points for the surgeon to consider in the use of caudal anesthesia are:

One needle injection is simpler.

The more needles used the more likelihood of entering the sacral venous plexus and the spinal canal.

The minimum amount of pain to the patient.

The lessening of the possibility of slough following injection.

The only disadvantage encountered is the time that must be allowed to elapse before the anesthesia is complete.

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SIXTH DISTRICT—O. O. FEASTER, M.D. . . . *St. Petersburg*
Pinellas.

SEVENTH DISTRICT—J. RALSTON WELLS, M.D. . *Daytona Beach*
Brevard, Volusia, Seminole.

EIGHTH DISTRICT—S. D. RICE, M.D. . . . . *Gainesville*
Putnam, Levy, Baker, Bradford, Union, Flagler, Alachua.

NINTH DISTRICT—J. M. NIXON, M.D. . . . . *Panama City*
Holmes, Washington, Bay.

TENTH DISTRICT—G. C. OVERSTREET, M.D. . . . *Lakeland*
Polk.

ELEVENTH DISTRICT—M. J. FLIPSE, M.D. . . . . *Miami*
Dade.

TWELFTH DISTRICT—W. H. GRACE, M.D. . . . *Ft. Myers*
Glades, Charlotte, Hendry, Lee, Collier.

THIRTEENTH DISTRICT—A. M. C. JOBSON, M.D. . . *Tampa*
Hillsboro, Hernando, Pasco.

FOURTEENTH DISTRICT—D. A. MCKINNON, M.D. . *Marianna*
Calhoun, Jackson, Gulf.

FIFTEENTH DISTRICT—C. W. SHACKELFORD, M.D.
Palm Beach, Broward . . . . . *West Palm Beach*

SIXTEENTH DISTRICT—S. C. WOOD, M.D. . . . *Leesburg*
Sumter, Lake.

SEVENTEENTH DISTRICT—SPENCER A. FOLSOM, M.D. . *Orlando*
Osceola, Orange.

EIGHTEENTH DISTRICT—H. GATES, M.D. . . . . *Bradenton*
Manatee, Sarasota.

NINETEENTH DISTRICT—HENRY P. BEVIS, M.D. . . . *Arcadia*
DeSoto, Hardee, Highlands.

TWENTIETH DISTRICT—WILLIAM R. WARREN, M.D. . *Key West*
Monroe.

TWENTY-FIRST DISTRICT—H. D. CLARK, M.D. . *Ft. Pierce*
St. Lucie, Okeechobee, Indian River, Martin.

PRESIDENT'S SPECIAL APPOINTMENTS

REPRESENTATIVE, FLORIDA HEALTH COUNCIL
H. MASON SMITH, M.D. . . . . *Tampa*

ADVISORY COMMITTEE TO WOMAN'S AUXILIARY
FREDERICK J. WAAS, M.D. . . . . *Jacksonville*
J. H. PIERPONT, M.D. . . . . *Pensacola*
J. E. TAYLOR, M.D. . . . . *DeLand*
J. S. MCEWAN, M.D. . . . . *Orlando*
W. W. MASSEY, M.D. . . . . *Quincy*

GET WHAT IT'S WORTH

Merchants in a city do everything to get business. They keep up the appearance of the store, they stock good quality merchandise and they advertise.

Physicians are merchants in a certain sense of the word. They have to make a living with perhaps enough in the savings to guard against the inevitable time when the hand is no longer steady and the eye grown dim. They modernize their office, give their patients their best and get advertising through these pleased persons.

It is true that in medicine there is no set scale of prices. But there is an average fee in a community. Some doctors, not all but some, are cutting below these average fees to attract a volume of business and keep others from getting these cases. People, as a rule, know doctors only by reference of physician or friend. Some people shop. When one doctor lowers his fees, he gets

the shoppers. But soon the others get on to this and come down to the level of the lowered fee to meet competition and keep clientele. As a result, the first to cut, must cut again or he is right back where he started. The public is pleased by the "price war", and the physicians suffer loss in income.

An ethical practitioner should not belittle his work by charging less than the established fee. He should tell the shoppers the regular fee and stick to it. If the case is worthy of a lowered fee, or even charity, no one would refuse. But the day is not far off when the medical profession will be bossed by the laity instead of being respected and honored if our business methods do not improve. Competition is fine when it is clean and just. We do not want to fall in line with the cut raters.

COUNTY SOCIETIES

Each county society is a component part of the State Association which is in turn a part of the National Association. The county society, therefore, is the representative of the state and national groups and is in touch with the laity more closely than the other organizations, therefore the actions of each county society and the members of the society are judged by the public and reflected for or against the state and national organizations.

The actions of the county societies are ofttimes carefully scrutinized by the public more so than we realize, quite frequently doctors will hear the laymen make remarks about the illegitimate work of other doctors who are members of organized medicine. This is and should be embarrassing to the honest and conscientious man who knows that such work is being carried on by a fellow member of his society. It is the duty of every doctor to report the irregularities to the Board of Censors of his society and have an impartial investigation made of these men and if they are found guilty their connection with the local society should be suspended immediately. This will occasionally result in some criticism of the person who started the investigation but in the long run will be of benefit to the individual and to organized medicine as a whole. The sooner the laity discovers that organized medicine will not permit irregular or illegitimate work by its members the more respect they will have for organized medicine.

This is the duty of the county society and should be supported and encouraged by the State and National Associations.

One county society in this state during the past year has suspended relations with five members, two for conduct unbecoming a gentleman and a physician and three for other causes. This society should be commended for its stand and particularly its few members who undertook these investigations and demanded that this action be taken.

It might be well for other local societies to investigate their ranks and take a definite stand against irregularities of any kind by its members, for judgment of organized medicine by the public depends to a great extent upon the action of local county societies and their members.

STATE NEWS ITEMS

The American College of Physicians will hold its fifteenth annual clinical session at Baltimore, Maryland, from March 23rd to 27th, inclusive, 1931. The Lord Baltimore Hotel will be headquarters. Dr. Sydney R. Miller, Baltimore, as president, will have charge of the selection of the general scientific program. Dr. Maurice C. Pincoffs of Baltimore, has been appointed by the Board of Regents as the General Chairman of the Session, and will make all local arrangements, including the making up of the program of clinics. Business details will be handled by the Executive Secretary, Mr. E. R. Loveland, from the college headquarters, 133-135 S. 36th St., Philadelphia.

* * *

Dr. G. H. Edwards of Orlando recently spent a three-weeks vacation in the north. He attended the Kiwanis convention in Atlantic City as well as clinics in New York City.

* * *

Dr. L. M. Gable of St. Petersburg is at summer training camp at Fort Scriven, Ga. He is a lieutenant colonel of the Medical Reserve Corps.

* * *

Mr. and Mrs. Hobart Clark Hare announce the marriage of their daughter Helen to Dr. Shaler Arnold Richardson. The ceremony was quietly performed at 10 o'clock Friday morning, June 20, in the Church of the Good Shepherd by the Reverend C. A. Ashby, rector of the church. Dr. and Mrs. Richardson left immediately following the ceremony for a trip of two weeks' duration in the North.

Affiliation with the American Heart Association was decided upon at the first meeting of the executive committee of the Florida Heart Association, held in Jacksonville recently.

A tentative program was prepared to be carried out in conjunction with the Florida State Board of Health, Florida Medical Association and Florida Public Health Association.

The Florida Heart Association is only a little more than a year old, having been organized at St. Augustine, April 2, 1929. Its object is the study and prevention of heart diseases and its activities will include clinics, research and publicity and other forms of educational work relating to the cause, prevention and treatment of heart diseases.

Those present at the meeting were: Dr. Herrman H. Harris, vice-president and chairman of the scientific section; Dr. Louie Limbaugh, secretary-treasurer; Dr. T. Z. Cason, president of the Florida Public Health Association; Dr. Henry Hanson, state health officer, and Sherwood H. Smith, executive secretary of the Florida Public Health Association, all of Jacksonville.

* * *

Dr. C. M. Sandusky was appointed as an associate in the ear, nose and throat service of the medical staff at the Duval county hospital at the meeting of the Duval county welfare board recently. The appointment was made upon the recommendation of Dr. H. Marshall Taylor, chief of the service.

* * *

Dr. B. C. Wilson of Jacksonville, district medical officer of the State Board of Health for northeastern Florida, left recently for New York and Baltimore, where he is to take post-graduate work in public health work. Mrs. Wilson and daughter accompanied him. Dr. Wilson will return to Jacksonville about September 1.

* * *

Dr. Spencer Folsom of Orlando and Miss Mary Margaret Taylor of Beaver, Pa., were married on June 14th at Beaver, Pa.

* * *

Dr. T. H. Green recently moved from St. Petersburg to Nashville, Tenn.

* * *

Dr. H. D. Smith and family of Sanford spent a two-weeks vacation recently with friends and relatives at Slocum, Ala.

Dr. John Bell of Pensacola recently returned from St. Louis, Mo., where he attended clinics.

* * *

Friends of Dr. L. M. Anderson of Lake City will be interested in his recent appointment as Surgeon General on the staff of General Ayers, Commander of the Florida Division, United Confederate Veterans. This appointment carries with it the rank of colonel.

* * *

Dr. T. B. Echard of St. Petersburg has gone to Connellsville, Pa., his old home, for the summer.

* * *

The Escambia County Medical Society and the Staff of the Pensacola Hospital adjourned for the summer months at the June meeting.

* * *

Dr. J. S. McEwan of Orlando recently returned from attending his class reunion at Northwestern University. Dr. McEwan also attended clinics in Chicago while on this trip.

* * *

The Florida Society of Dermatology and Syphilology held a very interesting meeting recently at the office of Dr. J. L. Kirby-Smith of Jacksonville. As chairman of the Society, Dr. Kirby-Smith presided. The morning session was devoted to the presentation of cases and a discussion of rare skin diseases and their treatment. At the afternoon session, the business of the society was taken up. Dr. C. A. Andrews of Tampa was elected chairman and Dr. J. Frank Wilson of Jacksonville, secretary. Attending the meeting were Dr. C. A. Andrews and Dr. J. J. Saxon of Tampa; Dr. Elmo D. French, Miami; Dr. J. L. Kirby-Smith, Dr. T. A. Blinn and Dr. J. Frank Wilson of Jacksonville.

* * *

A baby girl was born to Dr. and Mrs. Corbett E. Tumlin, Miami, on May 2, 1930.

* * *

The many friends of Dr. C. Carroll of Apopka will regret to learn that he has for some time been ill at the Orange General Hospital, Orlando.

* * *

Dr. J. S. McEwan and Dr. Sylvan McElroy of Orlando recently returned from a fishing trip at Sebastian.

Dr. R. H. Knowlton recently returned from a two-weeks vacation, which he spent golfing in North Carolina.

* * *

As the full time County Health Service develops in Florida under the leadership of the State Board of Health, there will be need for a number of well-grounded physicians from 30 to 40 years of age, with not less than five years' experience in private practice, to serve as County Health Officers. Preference will be given to graduates of high grade southern medical colleges. Funds will be provided with which to give these men a special course of training placing them at the head of a unit. Only energetic men physically fit and of good character will be considered. Those interested may communicate with Dr. F. A. Brink, P. O. Box 4479, Jacksonville, Florida.

* * *

The licenses of six Florida physicians were revoked by the State Board of Medical Examiners in executive session June 16, at Lakeland. J. R. Vinson, Callahan; Robert Bruce McFeeters, Dowling Park; Horace J. Williams, Tampa, and M. P. Sporman, Manatee, were convicted of violation of the Harrison narcotic act; George A. Munch, Tampa, using the mails to defraud in selling bogus diplomas and licenses, and C. Wade Page, Chipley, stealing an automobile.

* * *

At the annual meeting of the American Medical Editors' and Authors' Association, held in Detroit, June 24th, Dr. Shaler Richardson was elected to serve on the Board of Governors of that organization.

* * *

Dr. and Mrs. Frank Gray are happy over the arrival of a baby girl at the Orange General Hospital, Orlando.

* * *

Dr. Will Wood has moved his office from Pine Castle to Holopaw, Florida.

* * *

Mrs. Frederick J. Waas of Jacksonville was recently re-elected president of the St. Vincent's Hospital Woman's Auxiliary. The other officers elected were: Mrs. Julian Gammon, vice-president; Mrs. Joseph Chilli, secretary, and Mrs. Howard E. Cochran, treasurer.

* * *

Applications for associate bacteriologist (medical) must be on file with the Civil Service Commission at Washington, D. C., not later than July 30, 1930.

The examination is to fill a vacancy in the position of Clinical Laboratorian, U. S. Veterans' Bureau Hospital, Palo Alto, Calif., and vacancies occurring in positions requiring similar qualifications throughout the United States.

The entrance salaries range from $3,200 to $3,700 a year. Higher salaried positions are filled through promotion.

* * *

Dr. C. A. Williams of St. Petersburg has gone to New England for the summer.

* * *

Five nurses recently graduated from the Riverside Hospital Training School, Jacksonville, and were presented with diplomas at commencement exercises held in the Woman's Club building. Dr. James D. Pasco, hospital physician, made the presentation to Genevieve McKinley, Jacksonville; Marion H. Dunham, Griffin, Ga.; Maeola A. Byrd, Griffin; Nellie Mae Roberts, Palatka; and Mary K. Middleton, Hastings. An interesting feature of the exercises was an address by Dr. Henry Hanson, State Health Officer, Jacksonville. Dr. Edward Jelks, attending physician at the hospital, presided.

* * *

Dr. Annette Bieker of St. Petersburg is spending the summer in Tennessee.

* * *

Dr. C. P. Bullard recently moved his offices from 215 Seybold Building to 2930 N.W. 8th Avenue, Miami.

* * *

Dr. Louis M. Orr of Orlando recently returned from a trip to New York where he attended the meeting of the American Urological Society.

* * *

Dr. E. A. Heibner of St. Petersburg spent the month of June at his old home in Kansas.

* * *

Dr. S. A. Clark of Lakeland returned in June from New York City, where he spent several weeks taking post-graduate work at the New York Lying-In Hospital and the Post-Graduate Medical School.

* * *

Mrs. T. W. Causey, wife of Dr. T. W. Causey of Lakeland, saved the lives of her two small boys recently while they were swimming in Lake Morton, when the older boy stepped into a hole and pulled the smaller boy in with him. Mrs. Causey, who was sitting on the shore, jumped into the lake to their rescue. The younger was unconscious when she reached him. The boys have fully recovered.

Dr. George E. Beckman was appointed as associate in the section of anesthetics of the Duval County Hospital medical staff at the meeting of the Duval County Welfare Board held in June. The appointment was made on recommendation of Dr. Gaston Day, chief of the section.

* * *

Miss Dorothy Driver, daughter of Mr. and Mrs. C. W. Driver, of 215 Beach Place, Tampa, became the bride of Dr. Joseph C. Inman, of Chattahoochee, at the home of Mr. and Mrs. Driver July 2, 1930.

The marriage ceremony was performed by the bride's cousin, Rev. W. J. Carpenter, of the First Methodist church of Largo, in the presence of relatives and intimate friends.

Dr. and Mrs. Inman left by motor car for New Orleans. They will be at home after July 15 at Chattahoochee, where Dr. Inman is a member of the medical staff of the state hospital.

* * *

Dr. C. C. Collins of Jacksonville recently returned after spending some time in the New England states, Canada, and Detroit, Mich., where he attended the meeting of the American Medical Association. He was accompanied on the trip by Mrs. Collins and his daughter, Frances, who are remaining in Waynesville, N. C., for several weeks.

* * *

Lieutenant-Colonel Raymond Sanderson of Jacksonville, has been ordered by the war department to report at Brooksfield, Texas, for a month's active duty in the medical corps. Lieutenant-Colonel Sanderson will rank from date of May 17, 1930, and is to be relieved from duty by August 2, to return to his home.

* * *

Dr. and Mrs. Gordon H. Ira of Jacksonville are enjoying a vacation trip in the north. Dr. Ira attended the annual meeting of the American Medical Association held in Detroit, Michigan, June 24-27. Mrs. Ira, as a delegate from Florida, attended the annual meeting of the Woman's Auxiliary of the American Medical Association. Before returning home, Dr. and Mrs. Ira will spend two weeks visiting points on the great lakes and in Minnesota and two weeks in the Berkshire Hills in Massachusetts.

* * *

The many friends of Dr. O. W. Gardner of Greensboro will regret to learn of the death of his father, which occurred recently at Camilla.

PROCEEDINGS OF THE ELEVENTH ANNUAL MEETING OF THE FLORIDA RAILWAY SURGEONS' ASSOCIATION

The Eleventh Annual Meeting of the Florida Railway Surgeons' Association was held at the San Carlos Hotel, Pensacola, May 5, 1930.

The meeting was called to order at 2 p. m. by J. H. Pierpont, M.D., chairman of the local committee on arrangements. The invocation was delivered by the Reverend G. H. Rousseau, of the First Baptist Church, Pensacola. The Association was welcomed to the city by Frank G. Renshaw, M.D., District Surgeon, L. & N. Ry., Pensacola, and a response on behalf of the Association was made by L. M. Anderson, M.D., Lake City. The annual presidential address was then delivered by Harold D. Van Schaick, M.D., Jacksonville, the subject being "The Responsibilities of the Railway Surgeon." R. A. Woolsey, M.D., Chief Surgeon, Frisco System, St. Louis, Mo., honor guest of the Association, presented an interesting discourse on "Intra-Spinal Block."

At the scientific meeting of the Association, the following papers were read and discussed:

"Non-Union of Fractures"—J. D. Bell, M.D., Pensacola.

"Gunshot Wound of the Tibia—Report of a Case"—Z. Brantley, M.D., Grandin.

"A Discussion of General Principles Underlying Diagnosis and Treatment of Fractures"—J. S. Turberville, M.D., Century.

The election of officers resulted as follows: Gaston H. Edwards, M.D., Orlando, president; Carol C. Webb, M.D., Pensacola, vice-president; E. W. Warren, M.D., Palatka, reelected secretary and treasurer.

The next meeting will be held in Orlando on the day preceding that of the meeting of the Florida Medical Association, according to a provision of the constitution.

The following committees have been appointed by the president to serve for the ensuing year:

SCIENTIFIC PROGRAM COMMITTEE

Thos. H. Bates, M.D., Chairman, Lake City.
J. Ralston Wells, M.D., Daytona Beach.
H. A. Leavitt, M.D., Miami.

EXECUTIVE COMMITTEE

M. M. Hannum, M.D., Chairman, Eustis.
Walter C. Page, M.D., Cocoa.

NECROLOGY COMMITTEE

Geo. C. Tillman, M.D., Chairman, Gainesville.
John D. Peabody, M.D., St. Petersburg.
Leon A. Peek, M.D., W. Palm Beach.

COUNTY SOCIETY	SECRETARY	MEETINGS				Dues Paid.
		Date	Time	Place	Luncheon?	
Alachua	J. E. Maines, Jr., M.D., Gainesville.	2nd Tuesday	12:00 Noon	White House	Yes.	88%
Bay	Don S. Fraser, M.D., Panama City.					50%
Brevard	I. K. Hicks, M.D., Melbourne.	Varies		Varies		90%
Broward	Ralph Lingeman, M.D., Ft. Lauderdale.	2nd Tuesday	8:00 P.M.	Chamber of Commerce	No.	75%
Columbia	T. H. Bates, M.D., Lake City.	1st Monday.	7:30 P.M.	Blanche Hotel		64%
Dade	E. N. McKenzie, M.D., Miami.	1st Friday	8:30 P.M.	Club Room, Huntington Bldg.	Occasionally.	77%
DeSoto-Hardee-Highlands	H. V. Weems, M.D., Sebring.		8:00 P.M.	Varies	Yes.	87%
Duval	Kenneth A. Morris, M.D., Jacksonville.	1st Tuesday	8:15 P.M.	Chamber of Commerce Building	No.	68%
Escambia	J. M. Hoffman, M.D., Pensacola.	1st Tuesday	8:00 P.M.	Board of Health Building	No.	69%
Hamilton	J. R. Bruce, M.D., Jasper.					100%
Hillsboro	J. T. Cowart, M.D., Tampa.	1st and 3rd Tuesdays	8:00 P.M.	Tampa Municipal Hospital	No.	79%
Jackson	C. H. Harrison, M.D., Cottondale.	2nd Tuesday	3:00 P.M.	Marianna	No.	64%
Lake	W. L. Ashton, M.D., Umatilla.	1st Thursday	12:30 P.M.	Eustis	Yes	100%
Lee	H. Quillian Jones, M.D., Ft. Myers.	3rd Friday	7:30 P.M.	Lee Memorial Hospital	No.	93%
Leon-Gadsden-Liberty-Wakulla-Jefferson	J. B. Brinson, Jr., M.D., Monticello.	Quarterly	3:00 P.M.	Varies	Yes.	85%
Madison	Geo. O. Davis, M.D., Madison.					
Manatee	A. Q. English, M.D., Manatee.	1st and 3rd Tues. Oct. to May; 2nd Tues. May to Oct.	7:00 P.M.	Dixie Grande Hotel	Yes.	92%
Marion	Thos. H. Wallis, M.D., Ocala.	3rd Thursday	12:30 P.M.	Harrington Hotel	Yes.	80%
Monroe	W. R. Warren, M.D., Key West.	1st Sunday	9:00 P.M.	Varies	Yes.	
Orange	J. R. Chappell, M.D., Orlando.	3rd Wednesday	8:30 P.M.	Varies	No.	67%
Palm Beach	R. G. Lewis, M.D., W. Palm Beach.	2nd Monday	8:00 P.M.	Court House	Yes.	66%
Pasco-Hernando-Citrus	Geo. R. Creekmore, M. D., Brooksville.	2nd Thursday	7:00 P.M.	Varies	Yes.	92%
Pinellas	O. O. Feaster, M.D., St. Petersburg.	Every other Friday	8:00 P.M.	500 Power & Light Bldg.	No.	69%
Polk	Herman Watson, M.D., Lakeland.	2nd Wednesday in Feb., Apr., June, Aug., Oct., Dec.	1:00 P.M.	Lakeland	Yes.	94%
Putnam	E. W. Warren, M.D., Palatka.	2nd Thursday	7:00 P.M.	James Hotel, Palatka	Yes.	60%
St. Johns	A. C. Walkup, M.D., St. Augustine.	3rd Tuesday	8:30 P.M.	Varies	Yes.	85%
St. Lucie-Okeechobee-Indian River-Martin	C. L. Davis, M.D., Okeechobee.	3rd Thursday	8:00 P.M.	Varies	Yes.	100%
Sarasota	F. C. Metzger, M.D., Sarasota.	2nd Tuesday	8:30 P.M.	Varies	Occasionally.	75%
Seminole	J. T. Denton, M.D., Sanford.	2nd Friday	8:00 P.M.	City Hospital		100%
Sumter	W. E. Mitchell, M.D., Coleman.	2nd Tuesday		Varies	No.	
Suwannee	W. C. White, M.D., Live Oak.					67%
Taylor	R. J. Greene, M.D., Perry.	Last Thursday	12:15 P.M.	Eldorado Cafe	Yes.	60%
Volusia	J. Ralston Wells, M.D.,				Yes.	78%

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Dade	E. N. McKenzie, M.D., Miami.	1st Friday	8:30 P.M.	Club Room, Huntington Bldg.	Occasionally.	77%
DeSoto-Hardee-Highlands	H. V. Weems, M.D., Sebring.		8:00 P.M.	Varies	Yes.	87%
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Lake	W. L. Ashton, M.D., Umatilla.	1st Thursday	12:30 P.M.	Eustis	Yes	100%
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Leon-Gadsden-Liberty-Wakulla-Jefferson	J. B. Brinson, Jr., M.D., Monticello.	Quarterly	3:00 P.M.	Varies	Yes.	85%
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Monroe	W. R. Warren, M.D., Key West.	1st Sunday	9:00 P.M.	Varies	Yes.	
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Palm Beach	R. G. Lewis, M.D., W. Palm Beach.	2nd Monday	8:00 P.M.	Court House	Yes.	66%
Pasco-Hernando-Citrus	Geo. R. Creekmore, M. D., Brooksville.	2nd Thursday	7:00 P.M.	Varies	Yes.	92%
Pinellas	O. O. Feaster, M.D., St. Petersburg.	Every other Friday	8:00 P.M.	500 Power & Light Bldg.	No.	69%
Polk	Herman Watson, M.D., Lakeland.	2nd Wednesday in Feb., Apr., June, Aug., Oct., Dec.	1:00 P.M.	Lakeland	Yes.	94%
Putnam	E. W. Warren, M.D., Palatka.	2nd Thursday	7:00 P.M.	James Hotel, Palatka	Yes.	60%
St. Johns	A. C. Walkup, M.D., St. Augustine.	3rd Tuesday	8:30 P.M.	Varies	Yes.	85%
St. Lucie-Okeechobee-Indian River-Martin	C. L. Davis, M.D., Okeechobee.	3rd Thursday	8:00 P.M.	Varies	Yes.	100%
Sarasota	F. C. Metzger, M.D., Sarasota.	2nd Tuesday	8:30 P.M.	Varies	Occasionally.	75%
Seminole	J. T. Denton, M.D., Sanford	2nd Friday	8:00 P.M.	City Hospital		
Volusia	Daytona Beach.	2nd Tuesday	7:30 P.M.	Varies	Occasionally.	100%
Walton-Okaloosa	A. G. Williams, M.D., Lakewood.	3rd Thursday	8:00 P.M.	Varies		56%
Washington-Holmes	H. A. McClure, M.D., Chipley.					

NOTE—Secretaries: Please submit information to complete the above schedule.

THE WOMAN'S AUXILIARY PAGE

WOMAN'S AUXILIARY
TO THE
FLORIDA MEDICAL ASSOCIATION, INC.

State Editor
MRS. EDWARD JELKS,
2244 St. Johns Avenue
Jacksonville

OFFICERS

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MRS. S. E. DRISKELL, President-elect . . . . . *Jacksonville*
MRS. W. G. POST, JR., Vice-President . . . . . *St. Petersburg*
MRS. J. M. IRWIN, Historian . . . . . *St. Augustine*
MRS. J. E. TAYLOR, Secy.-Treas. . . . . *DeLand*

At the annual meeting of the Woman's Auxiliary of the American Medical Association, held in Detroit June 23-27, Florida's state organization was splendidly represented by our president, Mrs. J. Ralston Wells of Daytona Beach and two delegates, Mrs. Gordon H. Ira of Jacksonville and our past secretary-treasurer, Mrs. Rufus Thames of Milton. We are expecting excellent and inspiring reports from this meeting which we hope to publish in the August issue of the Journal.

COMMITTEES

The by-laws of the National Auxiliary ask that each State Auxiliary request the State Medical Society to appoint a committee of five members to act as an advisory committee to the State Auxiliary. Dr. Davis has appointed the following committee:

Dr. Frederick J. Waas, Chairman, Jacksonville.
Dr. J. Harris Pierpont, Pensacola.
Dr. J. E. Taylor, DeLand.
Dr. J. S. McEwan, Orlando.
Dr. W. W. Massey, Quincy.

Mrs. Wells announces the appointment of a committee on Revision of the Constitution and By-laws:

Mrs. J. J. Spencer, Chairman, St. Augustine.
Mrs. M. A. Lischkoff, Pensacola.
Mrs. H. C. Dozier, Ocala.

HYGEIA

Mrs. Herrman Harris, chairman of this committee, is striving to have as many health programs as possible throughout the state this year. This month she has the following to report: On June 5th, Dr. Upchurch, City Health Officer of Jacksonville, spoke to the Duval County Auxiliary. At the last meeting of the Parent-Teachers' Association of the Panama and Brentwood Schools, Dr. Harris spoke on the "Early Diagnosis of Tuberculosis."

VOLUSIA COUNTY

The Volusia County Medical Society and the Woman's Auxiliary closed a successful year with their annual picnic at DeLeon Springs. The supper which was arranged by the Auxiliary was served cafeteria style. There was a good attendance of members and their families and everybody seemed to enjoy the outing.

DUVAL COUNTY

The Duval County Auxiliary held its last meeting until next October on June 5th at the Jacksonville Chamber of Commerce building. The two outstanding features of this meeting were an excellent reading by Mrs. W. S. Manning of a sketch of the life of Dr. Alvan Wentworth Chapman and a talk on health by Dr. N. A. Upchurch, City Health Officer. The article on Dr. Chapman was written by Miss Winfred Kimball of Apalachicola and published by the New York Botanical Journal in 1921, Vol. 22, No. 253. Dr. Chapman practiced medicine many years at Apalachicola and was at the same time a great botanist. He was among the first to make a study of the flora of that part of Florida and was the first botanist to discover and classify the rhododendron which is found growing in that part of the state. Grey, the famous botanist, who was friend of Chapman's and visited him at Apalachicola, named this specie "Rhododendron Chapman" in honor of its discoverer.

The talk on Health, by Dr. Upchurch, was most enlightening and instructive. Few present had realized the scope of the work of the City Health Department.

In the report of the meeting at Pensacola, the Duval County Auxiliary was particularly proud and also deeply appreciative of the honor accorded it at the state meeting where its president, Mrs. S. E. Driskell, was elected president of the State Association for 1931.

Summer Diarrhea

The following formula provides a means of supplying the principal fuel utilized in the body for the production of heat and energy and furnishes immediately available nutrition well suited to protect the proteins of the body, to prevent rapid loss of weight, to resist the activity of putrefactive bacteria, and to favor a retention of fluids and salts in the body tissues:

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TUBERCULOSIS ABSTRACTS

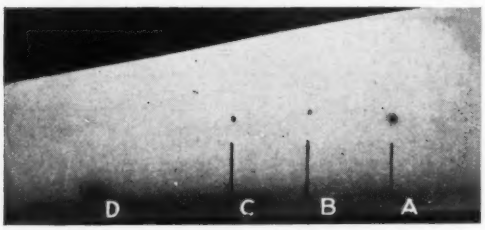
A REVIEW FOR PHYSICIANS

ISSUED MONTHLY BY THE NATIONAL TUBERCULOSIS ASSOCIATION

Early European statistics, based largely on studies of groups suspected of having tuberculosis and living in populous cities, created the impression that all adults and nearly all children reacted positively to the tuberculin test. For that reason, the value of the test was unjustly discounted. Infection is not so prevalent in this country, especially among children, and since the tuberculin reaction is a very simple measure and furnishes a base line in the diagnosis of early tuberculosis, its value is being re-established. Charles Hendee Smith, from whose paper, "Tuberculin Skin Reactions," the following abstracts are derived, pleads for a more consistent use of the test, and at the same time emphasizes the need for greater precision in making and interpreting the test.

TUBERCULIN SKIN REACTIONS

Tissues which have once been sensitized by living tubercle bacilli develop the power to react to tuberculin. A positive tuberculin reaction means



Pirquet Reactions and Control
A—Pirquet positive, B—Control
C—Pirquet negative, D—Mantoux

that the tubercle bacillus has lived and grown in the body. Koch introduced the general, or subcutaneous tuberculin reaction in 1890. In 1907, Pirquet developed the cutaneous scratch test, and the same year Wolff-Eisner and Calmette described the conjunctival test, while Moro gave us the percutaneous or inunction test. About a year

(Continued on page 42)

Index to Advertisements

THIS ISSUE

American Optical Co. 7
Arcade Prescription Pharmacy 47
Atlanta Neurological Hospital 46
Attwood, J. K., Pharmacist 42
Aznoe's Natl. Physicians' Exchange 46

Brawner's Sanitarium 44
Bunce, Landham & Klugh, Drs. 39

Camp & Co., S. H. 47
Combs Funeral Homes (Ambulance) 47
Conant Co., Marcus (Ambulance) 47

Ferguson Undertaking Co. (Ambulance) 47

Glenwood Park Sanitarium 42
Guyer X-ray Co. ..... Second Cover

Hand, Carey (Ambulance) 47
Hart & Co., Ltd., E. J. 8
Hynson-Westcott & Dunning, Inc. 39

Jones, William D., Pharmacist 46

Knox Gelatine Laboratories 9

Laboratories—Drs. Lake and Ayers 8
Laboratory Products Co., The ..... Third Cover
Lederle Laboratories ..... Back Cover
Lilly, Eli & Co. 5, 6, 10
Lister Bros., Inc. 8

Maltbie Chemical Co., The ..... Front Cover
Mead Johnson & Co. 45
Mellin's Food Co. 39
Merck & Co., Inc. 48
Miami Retreat 42
Moulton & Kyle (Ambulance) 47

Nonspi Co., The 44

Parke, Davis & Co. 43

Record Co., The 48

Squibb & Sons, E. R. 41
Storm, M.D., Katherine L. 8
Sugar Institute, The 3
Surgical Supply Co. 4

Tucker Sanatorium, Inc. 4

Veil Maternity Hospital 4

Wallace Sanitarium 8

Summer Outdoor Activity Greatly Increases Tetanus Cases

TETANUS occurs in every month of the year, but the danger is greatly increased during the warm months. Outdoor activity increases exposure to injury, and thus increases the number of burns, lacerations, punctures from splinters and nails, and cuts, abrasions and fractures. Exposure of any wound to street dirt or garden soil may be followed by tetanus infection. The burns which children sustain from fireworks during Fourth of July celebrations also cause many cases of tetanus.

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later, Mantoux introduced the intracutaneous test, and shortly after that Hamburger and Monti used the hypodermic, or "Stich" method. The object of each of these tests is to bring tuberculin into contact with the deeper epidermal cells and, therefore, the intracutaneous and "Stich" tests may be expected to be more accurate than the Pirquet and Moro tests.

PIRQUET TEST UNRELIABLE

Because of the simplicity of the technique, the Pirquet test has gained popularity and has been used in many countries as a routine procedure. But it has distinct disadvantages. If the scratch is too deep, the subsequent swelling may, to an untrained observer, resemble a positive reaction. A greater disadvantage is the uncertainty of the test because the tuberculin may not be kept in contact with the cells of the deeper layer of the skin long enough to excite the specific response. Many children fail to react on the first test, yet show a positive reaction when retested a few days later. This is not due to the raising of the sensitivity by the first test, for if two tests are made at the same time, one may fail while the other reacts sharply.

(Continued on Page 44)

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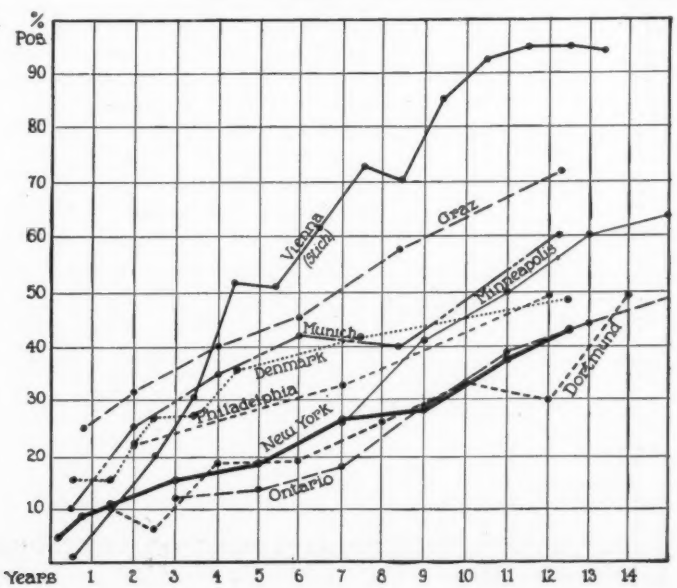

Curves marked with names of cities showing incidence of positive Mantoux tests by years.

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The unreliability of the Pirquet test, says Smith, became manifest to those in the Children's Medical Division at Bellevue Hospital about ten years ago. The Mantoux reaction was adopted in the hope of finding a more accurate test. Immediately, it was found that many patients reacted definitely to the Mantoux test and failed to react to the Pirquet, although two of the latter tests were always done. This difference persisted year after year, and at about the same rate; *i. e.*, the Mautoux test giving about twice as many positive reactions as the Pirquet. For several years, both tests were performed on all children, but the Pirquet was finally dropped as a waste of time because of its uncertainty.

CAREFUL TECHNIQUE ESSENTIAL

After trying various dilutions, Smith found that 1:1,000 is the most satisfactory, stronger solutions (1:200, 1:100, or 1:10) being used if necessary to retest, though these stronger solutions should never be used for the first test as sloughing may result. Making up and keeping the solution is not difficult, but the usual precautions must be strictly observed and errors of technique must be guarded against. For example, a needle or syringe that has been used for tuberculin should never be used for the control nor for a Schick test, as tuberculin may remain active after boiling several hours and cause a reaction. Acute illness, especially if there is a high fever, may suppress the reaction. Severe tuberculosis, such as general miliary tuberculosis or a wasting, chronic disease, may obscure the reaction. The tuberculin must not be too old nor contaminated with bacteria or molds.

PIRQUET AND MANTOUX TESTS COMPARED

As a means of comparing the reliability of the Pirquet with the Mantoux test, 3,112 children (ranging in age from birth to 13 years) were given two Pirquet tests at the same time that the Mantoux was given. Seven and eight tenths per cent reacted positively to the Pirquet test, while

(Continued on Page 46)

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16.5% responded to the Mantoux. This confirms the opinion of others that the Pirquet test is unreliable and that epidemiological studies based on the Pirquet test are, for that reason, open to criticism. Smith has gathered and compared graphically the tuberculin test results of various groups of children in American and European cities. All of these inscribe so characteristic a curve or slope that it is quite possible to calculate the incidence of positive reactors at given ages in a given place. It is interesting that the curves for Vienna and Graz are distinctly higher than for other European cities and that all the curves of American cities are of about the same incidence. Not more than 40 to 50 per cent in the large American cities harbor the bacillus at puberty.

It must be remembered, concludes Smith, that "the army of the tuberculous is recruited from those infected in childhood" and "phthisis is the last verse of a song the first of which was sung in the cradle." These infected children, then, who carry tubercle bacilli in their bronchial nodes or elsewhere, are the ones who need early, correct diagnosis. The Mantoux test provides the means of making it. It should be used on every child who is underweight, anemic or languid or who has an unexplained irregular fever. These children with "tuberculous infection," so-called, have true incipient tuberculosis, latent or active. This disease is generally in lymphatic tissue, where it is easily encapsulated and where scar tissue does no great harm. Under intelligent, watchful care they usually do well. When the disease has involved more important structures, such as the lungs, bones or meninges, the damage is greater, the cure is more difficult or impossible, and the diagnosis has been made too late.—*Tuberculin Skin Reactions, Charles Hendee Smith, Am. Jour. of Dis. of Children, Dec., 1929, Vol. 38.*

(This review secured by the Florida Public Health Association from the National Tuberculosis Association).

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